ASSESSMENT REPORT ON

FORMALDEHYDE

FOR DEVELOPING AMBIENT AIR QUALITY OBJECTIVES

Alberta Environment

ASSESSMENT REPORT ON
FORMALDEHYDE
FOR DEVELOPING AMBIENT AIR QUALITY OBJECTIVES

Prepared by
Toxico-Logic Consulting Inc.

for
Alberta Environment

February 2006

ISBN No. 978-0-7785-6802-5 (Printed Version
ISBN No. 978-0-7785-6803-2 (On-Line Version)
Web Site: http://www.environment.alberta.ca/

Although prepared with funding from Alberta Environment (AENV), the contents of this report/document do not necessarily reflect the views or policies of AENV, nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

Any comments, questions, or suggestions regarding the content of this document may be directed to:

Air Policy Branch
Alberta Environment
10th Floor, Oxbridge Place
9820 – 106th Street
Edmonton, Alberta T5K 2J6
Fax: (780) 422-4192

Additional copies of this document may be obtained by contacting:

Information Centre
Alberta Environment
Main Floor, Oxbridge Place
9820 – 106th Street
Edmonton, Alberta T5K 2J6
Phone: (780) 427-2700
Fax: (780) 422-4086
Email: env.infocent@gov.ab.ca

FOREWORD

Alberta Environment maintains Ambient Air Quality Objectives[1] to support air quality management in Alberta. Alberta Environment currently has ambient objectives for more than thirty substances and five related parameters. These objectives are periodically updated and new objectives are developed as required.

With the assistance of the Clean Air Strategic Alliance, a multi-stakeholder workshop was held in October 2004 to set Alberta's priorities for the next three years. Based on those recommendations to Alberta Environment, a three-year work plan was developed to review four existing objectives, and create three new objectives.

This document is one in a series of documents that presents the scientific assessment for these substances.

Laura Blair
Project Manager
Air Policy Branch

[1] **NOTE**: The *Environmental Protection and Enhancement Act*, Part 1, Section 14(1) refers to "ambient environmental quality objectives" and uses the term "guidelines" in Section 14(4) to refer to "procedures, practices and methods for monitoring, analysis and predictive assessment." For consistency with the *Act*, the historical term "ambient air quality guidelines" is being replaced by the term "ambient air quality objectives." This document was prepared as the change in usage was taking place. Consequently any occurrences of "air quality guideline" in an Alberta context should be read as "air quality objective."

ACKNOWLEDGEMENTS

The authors of this report would like to thank Laura Blair of Alberta Environment for inviting the submission of this assessment report. Toxico-Logic Consulting Inc. would like to acknowledge and thank the following authors who participated in the completion of this report.

Dr. Selma Guigard
Edmonton, Alberta

Dr. Warren Kindzierski
WBK & Associates Inc.
St. Albert, Alberta

Colleen Purtill
Toxico-Logic Consulting Inc.
Calgary, Alberta

Jason Schulz
Edmonton, Alberta

Deirdre Treissman
Treissman Environmental Consulting Inc.
Calgary, Alberta

Dr. John Vidmar
Edmonton, Alberta

TABLE OF CONTENTS

FOREWORD i
ACKNOWLEDGEMENTS ii
LIST OF TABLES v
ACRONYMS AND ABBREVIATIONS vi
SUMMARY viii

1.0 INTRODUCTION 1

2.0 GENERAL SUBSTANCE INFORMATION 2
2.1 Physical, Chemical and Biological Properties 2
2.2 Emission Sources and Ambient Levels 3
2.2.1 Natural Sources 4
2.2.2 Anthropogenic Sources 4
2.2.3 Ambient Levels 5

3.0 ATMOSPHERIC CHEMISTRY AND FATE 8

4.0 EFFECTS ON HUMANS AND ANIMALS 10
4.1 Overview of Chemical Disposition 10
4.2 Genotoxicity and Carcinogenicity 11
4.3 Acute and Subacute Effects 12
4.3.1 Acute and Subacute Human Effects 12
4.3.1.1 Respiratory and Ocular Effects 12
4.3.1.2 Other Effects 14
4.3.2 Acute and Subacute Animal Effects 14
4.3.2.1 Respiratory Effects 15
4.3.2.2 Other Effects 17
4.4 Chronic Effects 19
4.4.1 Chronic Human Effects 19
4.4.1.1 Respiratory Effects 28
4.4.1.2 Carcinogenic Effects 29
4.4.1.3 Other Effects 30
4.4.2 Subchronic and Chronic Animal Effects 31
4.4.2.1 Death 31
4.4.2.2 Respiratory Effects 31
4.4.2.3 Immunological/Lymphoreticular 39
4.4.2.4 Cancer 39
4.4.2.5 Other Effects 39

5.0 EFFECTS ON VEGETATION 40

6.0 EFFECT ON MATERIALS 42

7.0 AIR SAMPLING AND ANALYTICAL METHODS 43
7.1 Reference Methods 43

7.1.1 US EPA Compendium Method TO-5 43
7.1.2 US EPA Compendium Method TO-11A 43
7.1.3 US EPA Compendium Method TO-15A 44
7.1.4 NIOSH Method 2016 45
7.1.5 NIOSH Method 2541 45
7.1.6 NIOSH Method 3500 46
7.1.7 OSHA Method 52 46
7.1.8 OSHA Method ID-205 46
7.2 Alternative, Emerging Technologies 47
7.2.1 Chromatographic Techniques 47
7.2.2 Spectroscopic Techniques 48
7.2.3 Colorimetric Techniques 50
7.2.4 Fluorimetric Techniques 50
7.2.5 Chemiluminescent Techniques 51
7.2.6 Passive Techniques 51

8.0 AMBIENT OBJECTIVES IN OTHER JURISDICTIONS 55
8.1 Formaldehyde Air Quality Guidelines and Objectives 55
8.1.1 Canada 55
8.1.2 United States 55
8.1.3 International Agencies 56
8.2 Use of Occupational Limits for Ambient Air Quality Guidelines 56

9.0 REFERENCES 59

APPENDIX A 69

APPENDIX B 83

LIST OF TABLES

Table 1 Identification of Formaldehyde 2

Table 2 Physical and Chemical Properties of Formaldehyde 3

Table 3 Environmental Fate of Formaldehyde 9

Table 4 Total Formaldehyde Emissions in Alberta According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by total emissions) 6

Table 5 Formaldehyde Air Emissions in Alberta According to the 2003 NPRI Database (EC, 2005)(in tonnes, ranked by total emissions) 7

Table 6 NOAELs and LOAELs for Acute Formaldehyde Inhalation (Human) 13

Table 7 NOAELs and LOAELs for Acute Formaldehyde Inhalation (Experimental Animals) 16

Table 8 NOAELs and LOAELs for Subacute Formaldehyde Inhalation (Experimental Animals) 18

Table 9 NOAELs and LOAELs for Subchronic and Chronic Formaldehyde Inhalation (Human) 20

Table 10 Examples of Case-Controlled Studies of Formaldehyde Inhalation (Human) (EC, 2001 and WHO, 2002) 21

Table 11 Examples of Cohort Studies of Formaldehyde Inhalation (Human) (EC, 2001 and CICAD, 2002) 24

Table 12 NOAELs and LOAELs for Subchronic Formaldehyde Inhalation (Experimental Animals) 32

Table 13 NOAELs and LOAELs for Chronic Formaldehyde Inhalation (Experimental Animals) 35

Table 14 Advantages and Disadvantages of Sampling and Analytical Methods 53

Table 15 Summary of Ambient Air Quality Guidelines for Formaldehyde 58

ACRONYMS AND ABBREVIATIONS

ACGIH	American Conference of Governmental Industrial Hygienists
AENV	Alberta Environment
ATSDR	Agency for Toxic Substances and Disease Registry
Cal EPA	California Environmental Protection Agency
CI	Confidence Interval
DNPH	Dinitrophenylhydrazine
DNSH	Dansylhydrazine
DOAS	Differential Optical Absorption Spectroscopy
EC	Environment Canada
ECD	Electron Capture Detection
ESL	Effects Screening Level
FTIR	Fourier Transform Infrared Spectrometry
GC/FID/MS	Gas Chromatography/Flame Ionization or Mass Spectrometry Detection
HCHO	Formaldehyde (Chemical formula)
HPLC	High Performance Liquid Chromatography
hv	Energy
IARC	International Agency for Research on Cancer
IRIS	Integrated Risk Information System
LIFS	Laser-induces Fluorescence
LOAEL	Lowest Observable Adverse Effect Level
MRL	Minimum Risk Level
MW	Molecular Weight
NIST	National Institute of Standards and Technology
NIOSH	National Institute for Occupational Safety and Health
NOAEL	No Observable Adverse Effect Level
NPRI	National Pollutant Release Inventory
OECD	Organization for Economic Cooperation and Development
OR	Odds Ratio
OSHA	Occupational Safety and Health Administration
OME	Ontario Ministry of the Environment
PMR	Proportionate Mortality Ratio
REL	Reference Exposure Level
RR	Relative Risk
RsC	Risk Specific Concentration
SEER	Surveillance, Epidemiology, and End Results program
SIM	Select Ion Monitoring
SIR	Standardized Incidence Ratio
SMR	Standardized Mortality Ratio
SPIR	Standardized Proportionate Incidence Ratio
STEL	Short-Term Exposure Limit

RR	Relative Risk
TCD	Thermal Conductivity Detection
TDLAS	Tunable Diode Laser Absorption System
TEL	Threshold Effects Exposure Limit
TLV	Threshold Limit Value
US EPA	United States Environmental Protection Agency
UV/VIS	Ultraviolet/Visible
VOC	Volatile Organic Compound
WHO	World Health Organization

SUMMARY

Formaldehyde (HCHO) is a colorless, flammable gas with a pungent, suffocating odour. It is considered one of the most widely used industrial and research chemicals, used primarily in the manufacturing of resins, fertilizers and other compounds such as pentaerythriol, hexamethylenetetramine, artificial cellulose esters, dyes, urea, thiourea, organic chemicals, glass mirrors and explosives.

In air, formaldehyde has a short half-life due to reaction with sunlight (photolysis) and free radicals, specifically the photochemically produced hydroxyl radical (OH·). Reactions with other radicals and compounds such as nitrate radicals, hydroperoxyl radicals, hydrogen peroxide, ozone and chlorine play a minor role in the fate of formaldehyde in the atmosphere. Formaldehyde is highly water soluble, will transfer to atmospheric water and be removed by wet deposition, thereby limiting long-range transport.

Formaldehyde occurs naturally in the environment from combustion, biodegradation and photochemical decomposition of organic material. It is also produced as a result of the oxidation of naturally-occurring hydrocarbons (methane, terpenes and isoprene).

Anthropogenic sources of formaldehyde include combustion processes (may emit formaldehyde directly or emit the hydrocarbon precursors to formaldehyde), industrial processes and off-gassing of commercial materials and consumer products. Motor vehicles, power plants, incinerators, refineries, wood stoves, kerosene heaters, fireplaces, food cooking and cigarettes all lead to emissions of formaldehyde.

Formaldehyde emissions occur during formaldehyde production and the use of formaldehyde based fumigants, soil disinfectants, embalming fluids, and leather tanning agents. It is also emitted by the off-gassing of products or materials that contain formaldehyde or resins, such as particle board, plywood, wood-panels, furniture, urea-formaldehyde foam insulation, plastic surfaces, some varnishes, paints, coatings, wood preservatives, carpets, drapes, curtains, resin-treated fabrics, papers, disinfectants and sterilizing agents.

In Canada, anthropogenic emissions of formaldehyde are tracked by the Environment Canada National Pollutant Release Inventory program. In Alberta, the industrial sectors that release formaldehyde include the wood processing, non-metallic mineral products, pulp and paper, oil and gas, mining, chemical/chemical products, and primary metals sectors. Ambient concentrations of formaldehyde in the outdoor air of remote areas are generally <1 $\mu g\ m^{-3}$, concentrations in urban areas are typically <20 $\mu g\ m^{-3}$. Indoor air concentrations of formaldehyde can range from 20 to 60 $\mu g\ m^{-3}$.

In humans and animals, toxicity endpoints associated with acute inhalation of formaldehyde include irritation of the eye and upper respiratory tract as well as histological damage (at relatively high concentrations) to tissues in the upper respiratory tract. The lowest observable adverse effect levels (LOAELs) for these responses were 250 $\mu g\ m^{-3}$ in humans and >2,600 $\mu g\ m^{-3}$ in mice and rats. In guinea pigs, an increase in airway resistance was reported following 2

hours exposure to 380 μg m^{-3} formaldehyde (lowest LOAEL). An indication of compromised lung function (change in FEV_1>10%) was reported in humans following 1-hour exposure to approximately 3,700 μg m^{-3}.

Chronic (> 5 years) occupational exposure to formaldehyde (>300 μg m^{-3}) increased respiratory complaints (chronic bronchitis, shortness of breath, nasal irritation), eye irritation and histological damage to the nasal mucosa of exposed workers. Chronic exposure of animals to higher, possibly cytotoxic, air concentrations (>7,100 μg m^{-3}) resulted in substantial non-neoplastic changes in the upper airway (squamous metaplasia, basal hyperplasia, and rhinitis).

The majority of epidemiological studies of potential carcinogenic risk in humans indicate that there is no significant increase in risk of nasopharyngeal or lung cancer at relatively low exposure concentrations. Neoplastic effects (primarily nasal squamous cell carcinoma) were observed in rats at higher exposures (≥7,200 μg m^{-3}). Formaldehyde is weakly genotoxic; however, the results of the animal studies indicate that the carcinogenic response is dependant on cytotoxicity and cell proliferation as well as species physiology. The U.S. EPA reports sufficient evidence of data for carcinogenicity in animals and classifies formaldehyde as a *B1 - probable human carcinogen* (1991). An International Agency for Research on Cancer (IARC) Working Group in 2003 (IARC, 2004) recently reclassified formaldehyde as *carcinogenic to humans* (*Group 1*).

There were limited reports on the phytotoxicity of formaldehyde (fumigation) on plants; however, no significant effects were observed in a variety of plant species exposed to air concentrations above those causing human health effects (*i.e.*, > 400 μg m^{-3}). No significant effects were reported for the common Bean (*Phaseolus vulgaris*) exposed to up to 438 μg m^{-3} for 7 hours a day, 3 days a week over a 4 week period and no significant effects were reported for spinach (*Spinacia oleracea*), beets (*Beta vulgaris*), or oats (*Avena sativa*) exposed to up to 840 μg m^{-3} for 5 hours.

Standard air monitoring methods for formaldehyde are based on liquid impinger, coated-solid cartridge, canister, spectrometric, sorbent tube, or passive sampling approaches, followed by various analytical techniques. Widely employed and accepted reference air monitoring methods for formaldehyde have been developed, tested and reported by the United States Environmental Protection Agency (U.S. EPA), National Institute of Occupational Safety and Health (NIOSH), and Occupational Safety and Health Administration (OSHA). Emerging analyzing technologies for formaldehyde include: chromatographic, spectroscopic, colorimetric, fluorimetric, chemiluminescent, and passive techniques.

Ambient (outdoor) formaldehyde objectives have been developed by Alberta Environment (65 μg m^{-3} over 1-hour), the Ontario Ministry of the Environment (65 μg m^{-3} over 30 minutes at the maximum point of impingement and 65 μg m^{-3} over 24-hours), British Columbia MOE (60 μg m^{-3} as an action level and 370 μg m^{-3} as an episode level) and Manitoba Conservation (60 μg m^{-3} over 1-hour). The basis for the derivation of these air quality guidelines was, for the most part, unknown

The U.S. Agency for Toxic Substances and Disease Registry (ATSDR) established inhalation minimum risk levels (MRLs) for acute (1 to 14 days), intermediate (>14 to 364 days), and chronic (365 days and longer) exposure durations of 50 $\mu g\ m^{-3}$, 37 $\mu g\ m^{-3}$, and 10 $\mu g\ m^{-3}$, based on health effects data from human exposures and animal studies. The US EPA established an inhalation unit risk factor (1.3E-5 per $\mu g\ m^{-3}$) for formaldehyde based on the occurrence of squamous cell carcinomas in rats exposed over a lifetime.

Several U.S. agencies (Arizona, Massachusetts, Michigan, Minnesota, New Jersey, Rhode Island, Vermont, and Washington) have adopted or derived their 1-hour (in the case of Michigan) and annual average values from the US EPA inhalation unit risk factor. Only one agency (Arizona) used an occupational exposure limit (OSHA 15-minute short term exposure limit (STEL)) to develop a 1-hour ambient air guideline. Air quality criteria for 18 US states were reviewed. Formaldehyde guidelines ranged from 15 to 150 $\mu g\ m^{-3}$ over a 1-hour average, from 0.33 to 40 $\mu g\ m^{-3}$ over a 24-hour average and from 0.08 to 7.69 $\mu g\ m^{-3\ 3}$as an annual average.

1.0 INTRODUCTION

Ambient air quality objectives are established by Alberta Environment as part of the Alberta air quality management system, Section 14 of the Environmental Protection and Enhancement Act (AENV, 2000). The purpose of this assessment report was to provide a review of scientific and technical information to assist in evaluating the basis and background for an ambient air quality objective for formaldehyde. The following aspects were examined as part of the review:

- Physical and chemical properties;
- Existing and potential anthropogenic emissions sources in Alberta;
- Effects on humans, animals, and vegetation;
- Monitoring techniques, and;
- Ambient air guidelines and objectives in other jurisdictions.

The physical and chemical properties identified for formaldehyde include chemical structure, molecular weight, melting and boiling points, water solubility, density, vapour density, organic carbon partition coefficient, octanol water partition coefficient, vapour pressure, Henry's Law constant, bioconcentration factor, and odour threshold. A discussion of the behaviour of formaldehyde in the environment was also presented. Existing and potential natural and anthropogenic sources of formaldehyde emissions in Alberta were examined. Formaldehyde is a reportable substance on Environment Canada's National Pollutant Release Inventory.

Scientific information on the effects of formaldehyde on humans, animals, and vegetation were identified. Toxicity and epidemiology studies were located in peer-reviewed evaluations by Health Canada, the World Health Organization, the International Agency for Research on Cancer, and the ATSDR. The effects of formaldehyde on vegetation were identified following a comprehensive search of the Web of Science database. Data from the Canadian Environmental Protection Act Priority List Substance Assessment Report for Formaldehyde were also obtained.

Air sampling and analytical methods for formaldehyde used by regulatory agencies were included in this assessment. Standard air monitoring methods for formaldehyde employ liquid impinger, coated-solid cartridge, canister, spectrometric, sorbent tube, or passive sampling approaches, followed by various analytical techniques. Widely employed and accepted reference air monitoring methods for formaldehyde have been developed, tested and reported by the US EPA, NIOSH, and OSHA.

Ambient air guidelines for formaldehyde were established by a number of jurisdictions in North America for different averaging time periods. The majority of guidelines were developed using cancer risk assessment procedures and the US EPA inhalation unit risk factor for formaldehyde. The basis for how these approaches are used by different jurisdiction to develop guidelines was investigated in this report.

2.0 GENERAL SUBSTANCE INFORMATION

2.1 Physical, Chemical and Biological Properties

Formaldehyde (HCHO) is a colorless, flammable gas at room temperature (EC, 2001; ATSDR, 1999) with a pungent, suffocating odour (Genium, 1999; O'Neil, 2001). Formaldehyde is produced by catalytic vapour phase oxidation of methanol (O'Neil, 2001) and is considered one of the most widely used industrial and research chemicals (ATSDR, 1999). Formaldehyde is used primarily in the manufacturing of resins (EC, 2001; Genium, 1999), fertilizers (EC, 2001) and other compounds such as pentaerythriol, hexamethylenetetramine, artificial cellulose esters, dyes, urea, thiourea, organic chemicals, glass mirrors and explosives (Genium, 1999). It should be noted that pure formaldehyde is not available commercially (Lewis, 2000) since it polymerizes at low and ordinary temperatures (temperatures below 100°C) in the presence of polar compounds such as acids, alkalis or water (Reuss *et al.*, 2002). Formaldehyde is therefore sold as an aqueous solution (approximately 37 to 50% weight) containing methanol (Reuss *et al.*, 2000; Lewis, 2000) or other stabilizers (Reuss *et al.*, 2000).

The chemical formula, structure, registry numbers, synonyms, and trade names for formaldehyde are provided in Table 1 (NIST, 2003).

Table 1 Identification of Formaldehyde

Property	Value
Formula	CH_2O
Structure	O C H H
CAS Registry number	50-00-0
RTECS number	LP8925000
UN Number	UN1198 UN2209
Common Synonyms/Trade names	BFV; dormol; fannoform; formaldehyde solution; formaldehyde, gas; formalin; formalin 40; formalith; formic aldehyde; formol; fyde; HCHO; HOCH; Ivalon; Karsan; lysoform; methylaldehyde; methanal; methanal formalin; methyl aldehyde; methylene glycol; methylene oxide; morbicid; oxomethane; oxymethylene; paraform; polyoxymethylene glycols; superlysoform

The physical and chemical properties of formaldehyde are summarized in Table 2.

Table 2 Physical and Chemical Properties of Formaldehyde

Property	Value	Reference
Molecular Weight	30.026 g/mole	Lide, 2004
Physical state	colourless gas	Lide, 2004
Melting Point	-92 °C	Lide, 2004
Boiling Point	-19.1 °C	Lide, 2004
Density (liquid)	0.8153 at -20°C	Reuss *et al*., 2002
Density (gas) (air=1)	1.03	Verschueren, 2001
Vapour pressure	250 kPa at 20°C 516 kPa at 25°C 5.24 103 mm of Hg at 25°C	Verschueren, 2001 EC, 2001 Daubert and Danner, 1989[1]
Solubility in water	soluble in water $4.0\ 10^5$ mg L^{-1} at 20°C $5.5\ 10^5$ mg L^{-1} at 25°C	Lide, 2004 Pickrell *et al*., 1983[1] Amoore and Hautala, 1983[1]
Solubility	soluble in ethanol miscible in diethyl ether, acetone, benzene	Lide, 2004 Lide, 2004
pKa	13.27 at 25 °C	Lide, 2004
Henry's Law Constant	$3.27\ 10^{-7}$ atm $m^3.mol^{-1}$ $1.67\ 10^{-7}$ atm $m^3.mol^{-1}$ $3.36\ 10^{-7}$ atm $m^3.mol^{-1}$	ATSDR, 1999 Gaffney *et al*., 1987[1] Betterton and Hoffmann, 1988
Octanol water partition coefficient (log K_{ow})	0.35 -0.75	Lide, 2004 Verschueren, 2001
Organic carbon partition coefficient (log K_{oc})	0.70 to 1.57 1.57	EC, 2001 SRC, 1988[1]
Bioconcentration factor (log BCF)	0.036	SRC, 1988[1]
Flash Point	85 °C	Lide, 2004
Explosive limits	7.0 to 73%	Lide, 2004
Autoignition temperature	424 °C 300 °C	Lide, 2004
Odour threshold	0.5 to 1ppm (in air) 0.06 to 1.2 mg m^{-3}	ATSDR, 1999 Morandi and Maberti, 2001
Conversion factors for vapour (at 25 °C and 101.3 kPa)	1 mg m^{-3} = 0.815 ppm 1 ppm = 1.248 µg/m^3	Verschueren, 2001

[1]as cited in SRC, 2005

2.2 Emission Sources and Ambient Levels

Formaldehyde enters the environment from a number of natural and anthropogenic sources, but it is also produced as an intermediate in the oxidation of volatile organic compounds (EC, 2001) and hydrocarbons (ATSDR, 1999) in air. EC (2000) states that, although there are no reliable

estimates of the amount of formaldehyde produced directly by natural sources or indirectly by oxidation reactions, it can be expected that these amounts will be greater than those released from anthropogenic sources. EC (2000) notes; however, that despite the fact that natural sources and secondary production of formaldehyde are greater than anthropogenic sources, the highest formaldehyde concentrations have been measured near anthropogenic sources.

2.2.1 Natural Sources

Formaldehyde occurs naturally in the environment and results from combustion, biodegradation and photochemical decomposition of organic material (Reuss *et al.*, 2002). Examples of some natural combustion sources of formaldehyde include forest fires and brush fires (EC, 2001). Formaldehyde is also emitted during the decomposition of plant residues in soil (WHO, 1989). Formaldehyde is also produced as a result of the oxidation of naturally-occurring hydrocarbons, in particular methane and to a lesser extent, terpenes and isoprene, due to their shorter half-lives in the atmosphere (WHO, 1989).

2.2.2 Anthropogenic Sources

Anthropogenic emissions of formaldehyde result from combustion processes, industrial processes and off-gassing of commercial materials and consumer products (EC, 2001). Combustion processes account for most of the formaldehyde entering the atmosphere (ATSDR, 1999). Combustion processes may emit formaldehyde directly to the atmosphere or may emit hydrocarbons that will lead to the secondary formation of formaldehyde (ATSDR, 1999). Motor vehicles, power plants, incinerators, refineries, wood stoves, kerosene heaters, fireplaces, food cooking and cigarettes all lead to emissions of formaldehyde (ATSDR, 1999). Industrial processes and other activities that lead to formaldehyde emissions include venting during formaldehyde production, using formaldehyde as a fumigant, soil disinfectant, embalming fluid and leather tanning agent (ATSDR, 1999). Formaldehyde may also be emitted during the off-gassing of products or materials that contain formaldehyde or resins, such as particle board, plywood, wood-panels, furniture, urea-formaldehyde foam insulation (UFFI), plastic surfaces, some varnishes, paints, coatings, wood preservatives, carpets, drapes, curtains, resin-treated fabrics, papers, disinfectants and sterilizing agents (EC, 2001; ATSDR, 1999; WHO, 1989).

Industrial emissions of formaldehyde in Canada are provided in the 2003 National Pollutant Release Inventory (NPRI) database (EC, 2005). Tables 4 and 5 summarize formaldehyde emissions for Alberta: Table 4 summarizes the formaldehyde emissions to air, land and water and Table 5 provides details specifically related to air emissions of formaldehyde. Data for other Canadian provinces is presented in Appendix A. It should be noted that formaldehyde is also considered a volatile organic compound (VOC) and hence it is also included in the criteria air contaminants inventories of NPRI (EC, 2004).

The results in Tables 4 and 5 show that, in Alberta, the NPRI reported emissions of formaldehyde are almost exclusively to air, and that these air emissions are predominantly the result of stack or point source emissions. The industrial sectors that contribute to formaldehyde

emissions in Alberta are the wood industries sector, the non-metallic mineral products sector, the pulp and paper sector, the oil and gas sector, the mining sector, the chemical and chemical products sector and the primary metals sector.

2.2.3 Ambient Levels

Extensive ambient air concentration data for formaldehyde are presented in SRC (2005), EC (2000), IARC (1995), ATSDR (1999), Howard (1989) and WHO (1989); however no data specific to locations in Alberta were provided. The International Agency for Research on Cancer (IARC, 1995) reported that formaldehyde concentrations in the outdoor air in remote areas were typically below 0.001 mg m^{-3} while concentrations in urban areas were typically lower than 0.02 mg m^{-3}. The presence of formaldehyde in remote areas is believed to be due to its formation from the photooxidation of hydrocarbons transported to these remote areas (EC, 2001). In indoor air, formaldehyde concentrations typically range from 0.02 to 0.06 mg m^{-3}, although higher values have been measured in homes where urea-formaldehyde insulation, particle board or other formaldehyde-releasing materials have been used in the constructions of these homes (IARC, 1995). In recent years, indoor air formaldehyde concentrations have decreased due to a change in the construction materials used in homes (IARC, 1995).

Table 3 **Total Formaldehyde Emissions in Alberta According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by total emissions)**

NPRI ID	Company	City	Formaldehyde Emissions (in tonnes)			
			Air	Water	Land	Total
4880	Ainsworth Lumber Co. Ltd.	Grande Prairie	208.69	0	0	208.686
2762	Weyerhaeuser Canada Ltd.	Edson	40.744	0	0	40.744
2764	Weyerhaeuser Company Limited	Slave Lake	18.5	0	0	18.5
1251	Owens-Corning Canada Inc.	Edmonton	17.718	0	0	17.718
0001	Alberta Pacific Forest Industries	Boyle	10.86	0.14	0	11
3941	SOLEX Gas Processing Corp	Didsbury	10.36	0	0	10.36
5285	Apache Canada Limited	Zama City	10.1	0	0	10.1
4830	West Fraser Mills	Blue Ridge	10	0	0	10
6647	Albian Sands Energy Inc.	Ft. McMurray	7.368	0	0	7.368
15437	ATCO Gas	Carbon	7.254	0	0	7.254
3821	Canadian Fertilizers limited	Medicine Hat	7.192	0	0	7.192
1902	Nexen Canada Ltd.	Balzac	4.25	0	0	4.25
0011	Borden Chemical Canada, Inc.	Edmonton	3.829	0	0	3.829
6517	Footner Forest Products Ltd.	High Level	1.15	0	0	1.15
3269	Agrium Inc	Calgary	0.8	0	0	0.8
2963	Shell Chemicals Canada Ltd.	Fort Saskatchewan	0.626	0	0	0.626
5351	Baker Petrolite Corporation	Calgary	0	0	0	0.493
2316	Dow Chemical Canada Inc.	Red Deer	0.486	0	0	0.486
1162	Celanese Canada Inc.	Edmonton	0.345	0	0.001	0.346
0280	Dow Chemical Canada Incorporated	Fort Saskatchewan	0.056	0	0	0.056
6512	Norwood Foundry Ltd.	Nisku	0.011	0	0	0.011
7904	Target Products Ltd	Calgary	0	0	0	0.002
7905	Target Products Ltd	Mornville	0	0	0	0.002
0853	Marsulex Inc.	Fort Saskatchewan	0.002	0	0	0.002
2291	Brenntag Canada Inc. (AS65)	Calgary	0	0	0	0.001
2340	Univar Canada Ltd.	Calgary	0.001	0	0	0.001

Table 4 **Formaldehyde Air Emissions in Alberta According to the 2003 NPRI Database (EC, 2005)(in tonnes, ranked by total emissions)**

NPRI ID	Company	City	Formaldehyde Emissions (in tonnes)					
			Stack /Point	Storage /Handling	Fugitive	Spills	Other Non-Point	Total
4880	Ainsworth Lumber Co. Ltd.	Grande Prairie	208.686	0	0	0	0	208.69
2762	Weyerhaeuser Canada Ltd.	Edson	40.744	0	0	0	0	40.744
2764	Weyerhaeuser Company Limited	Slave Lake	18.5	0	0	0	0	18.5
1251	Owens-Corning Canada Inc.	Edmonton	17.7	0.018	0	0	0	17.718
0001	Alberta Pacific Forest Industries	Boyle	10.86	0	0	0	0	10.86
3941	SOLEX Gas Processing Corp	Didsbury	10.36	0	0	0	0	10.36
5285	Apache Canada Limited	Zama City	10.1	0	0	0	0	10.1
4830	West Fraser Mills	Blue Ridge	10	0	0	0	0	10
6647	Albian Sands Energy Inc.	Ft. McMurray	7.368	0	0	0	0	7.368
15437	ATCO Gas	Carbon	7.254	0	0	0	0	7.254
3821	Canadian Fertilizers limited	Medicine Hat	7.096	0	0.075	0	0.021	7.192
1902	Nexen Canada Ltd.	Balzac	4.25	0	0	0	0	4.25
0011	Borden Chemical Canada, Inc.	Edmonton	3.373	0.349	0.107	0	0	3.829
6517	Footner Forest Products Ltd.	High Level	1.15	0	0	0	0	1.15
3269	Agrium Inc	Calgary	0	0	0.8	0	0	0.8
2963	Shell Chemicals Canada Ltd.	Fort Saskatchewan	0.409	0.217	0	0	0	0.626
2316	Dow Chemical Canada Inc.	Red Deer	0.486	0	0	0	0	0.486
1162	Celanese Canada Inc.	Edmonton	0	0.241	0.104	0	0	0.345
0280	Dow Chemical Canada Incorporated	Fort Saskatchewan	0	0	0	0	0.056	0.056
6512	Norwood Foundry Ltd.	Nisku	0	0	0.011	0	0	0.011
0853	Marsulex Inc.	Fort Saskatchewan	0.002	0	0	0	0	0.002
2340	Univar Canada Ltd.	Calgary	0	0.001	0	0	0	0.001

3.0 ATMOSPHERIC CHEMISTRY AND FATE

The environmental fate of formaldehyde is presented by Howard (1989) and is summarized in Table 3. A detailed discussion is also presented in EC (2000) and ATSDR (1999).

In air, formaldehyde has a short half-life of a few hours due to its reaction with sunlight and free radicals (Larsen and Larsen, 1998). The primary reactions for formaldehyde in air are direct photolysis and reaction with photochemically produced hydroxyl radicals (OH·), the later being considered the most important (EC, 2001).

Photolysis of formaldehyde occurs according to one of the following two pathways (Atkinson, 1995):

$$HCHO + h\nu \longrightarrow \begin{matrix} \cdot H_2 + CO \\ \cdot H\cdot + H\dot{C}O \end{matrix}$$

where $h\nu$ represents energy. A lifetime ranging from 1.6 hours (Calvert *et al.*, 1972, as cited in EC, 2001) to 6 hours (Atkinson, 1995) has been proposed for formaldehyde based on these photolysis reactions.

Reaction of formaldehyde with the hydroxyl radical produces water and the HCO radical (Atkinson, 1995). This reaction and subsequent reactions with the HCO radical may lead to the production of water, carbon monoxide, formic acid and hydroperoxyl/formaldehyde adducts (Atkinson *et al.*, 1990, as cited in EC, 2001). The lifetime of formaldehyde due to the reaction with the hydroxyl radical has been estimated at 1.5 days (Atkinson, 1995). Half-lives in air range from 7.1 to 71.3 hours due to this reaction (Atkinson *et al.*, 1990, as cited in EC, 2001; Atkinson, 1985 as cited in EC, 2001).

Reactions of formaldehyde with other radicals and compounds such as nitrate radicals, hydroperoxyl radicals, hydrogen peroxide, ozone and chlorine are also possible; however, these are believed to play a minor role in the fate of formaldehyde in the atmosphere (EC, 2001).

Due to its high water solubility, it is expected that formaldehyde present in the air will transfer into atmospheric water: formaldehyde will be "washed out" of the air by clouds and precipitation and will be removed by wet deposition (EC, 2001). Although the data regarding the amount of formaldehyde removed by wet deposition varies greatly (see EC (2000)), it can be concluded that the half-life of formaldehyde in the troposphere will be shorter than predicted solely from the above reactions, since washout followed by wet deposition will also contribute to formaldehyde removal from air. As a result of its short half-life in the troposphere, it is expected that long-range transport of formaldehyde will be limited (EC, 2001; WHO, 1989).

Table 1 Environmental Fate of Formaldehyde

System	Fate	Reaction Rates
Water	• biodegrades to low levels within a few days • fate in groundwater is unknown • little sorption to sediment	• complete degradation of formaldehyde within 30 hours (under aerobic conditions) and 48 hours (under anaerobic conditions) in a stagnant lake
Soil	• aqueous solutions containing formaldehyde will enter soil • biodegradable of formaldehyde under aerobic and anaerobic conditions may occur • fate in soil is unknown	• no values reported
Air	• photolyzes and reacts rapidly with free radicals • reacts with nitrate radicals at night • due to formaldehydes high water solubility, it transfers to water in the atmosphere and surface water	• half-life of approximately 19 hours in clean air and 8 hours in polluted air • half-life in the troposphere during sunlight hours is of the order of a few hours • removal half-lives by dry and wet deposition are approximately 19 and 50 hours, respectively

4.0 EFFECTS ON HUMANS AND ANIMALS

Formaldehyde was listed as a priority chemical for risk assessment under the *Canadian Environmental Protection Act* (CEPA) by Health Canada and Environment Canada (as cited in EC, 2001). Formaldehyde is a normal product of animal metabolism and is naturally produced endogenously (OECD, 2002; Bolt, cited in ATSDR, 1999). It also has many industrial uses (chemical and plastics production, binders for wood products, etc.) and is product of forest fire emissions and fuel combustion (IARC, 2004; EC, 2001).

The focus of this section was adverse health effects associated with formaldehyde inhalation, oral and dermal effects were not reviewed in detail. Specific details of metabolic pathways and metabolic products are not described unless identified as significant in the assessment of health effects (*i.e.* reactive metabolites are produced). The primary literature sources for this review were the CEPA assessment report by Health Canada/Environment Canada (EC, 2001), the World Health Organization (WHO, 2002), IARC (2004), and the Agency for Toxic Substances and Disease Registry (ATSDR, 1999). The Organisation for Economic Development (OECD, 2002), the U.S. Environmental Protection Agency (on-line, latest revision 1991) and the WHO Environmental Health Criteria Document No. 89 (WHO, 1989) were also obtained, but not reviewed in detail. An on-line search of the scientific literature posted on TOXNET from 2001 to present was conducted to identify any information published since 2002; no additional documentation was identified.

4.1 Overview of Chemical Disposition

Formaldehyde is readily absorbed in the respiratory tract after inhalation. Absorption is thought to be almost 100% by the respiratory tissues (ATSDR, 1999). For nose-breathing animals (rats and mice) absorption occurs in the nasal cavity/mucosa (Casanova-Schmitz *et al.*, Casanova *et al.*, Heck *et al*, Heck *et al.*, Chang at al., cited in ATSDR, 1999). For oronasal breathers (primates, humans, dogs) absorption is expected to occur by the mucosa, trachea, and bronchi (ATSDR, 1999). Although fewer studies were available, absorption after ingestion was also estimated to be close to 100% (Burkhart *et al.*, Ells *et al.*, Galli *et al.*, Barry and Tome, Buckley *et al.*, cited in ATSDR, 1999). One study of liquid formaldehyde reported low (0.5%) dermal absorption in monkeys (Jeffcoat, cited in ATSDR, 1999).

Once absorbed into the blood after inhalation exposure, formaldehyde is very rapidly metabolized primarily by formaldehyde dehydrogenase using glutathione (GSH) into formate and CO_2 which are ultimately excreted via the kidneys and lungs (ATSDR, 1999; Casanova and Heck, Uotila and Koivusalo, cited in OECD, 2002). Metabolism is so quick that studies measuring blood formaldehyde concentrations did not report any increases in humans and dogs after inhalation (Heck *et al.*, Egle, cited in ATSDR, 1999 and OECD, 2002). Formaldehyde is also produced endogenously, in that it is a normal product of animal metabolism (OECD, 2002; Bolt, cited in ATSDR, 1999).

These results indicate that toxicity to internal organs would not be a factor with formaldehyde inhalation, only those tissues at the point of contact, specifically nasal and upper respiratory tract tissues and possibly lungs, with high exposures. This conclusion is supported by the human and animal studies of formaldehyde inhalation (See Sections 4.3 and 4.4 below).

The specific tissues affected are species dependant due to physical differences of the nasal passages (Schrieder, cited in ATSDR, 1999). In studies of rats, with radio-labelled formaldehyde, accumulation occurred in the respiratory and olfactory mucosa tissues with greater concentrations in the respiratory mucosa (Casanova-Schmitz *et al.*, cited in ATSDR, 1999). A similar study with monkeys reported the highest concentrations in the nasal tissue and the lowest concentrations in tissues of the upper part of the throat; two animals accumulated formaldehyde in the upper respiratory tract and lung tissues (Casanova *et al.*, cited in ATSDR, 1999).

The exact mechanism of adverse effects after exposure to exogenous formaldehyde (see Sections 4.3 and 4.4) is unknown. Formaldehyde is an irritant, corrosive, and is cytotoxic; high exposures produce degeneration and necrosis of the mucosal and epithelial layers (ATSDR, 1999).

4.2 Genotoxicity and Carcinogenicity

Formaldehyde was demonstrated to be weakly genotoxic in multiple *in-vitro* and *in-vivo* models (IARC, 2004). It has been reported to induce gene mutations and chromosomal aberrations in mammalian cells and produce DNA-protein cross links (DPCs) in animal and human studies (only in tissues with direct contact with formaldehyde) (IARC, 2004; OECD, 2002). Formaldehyde inhalation forms DNA-protein cross links (DPCs) in the nasal epithelium of rats (Casanova and Heck cited in OECD, 2002; Casanova-Schmitz, cited in ATSDR, 1999). Exposures greater than 3 ppm (3.75 mg m^{-3}) resulted in a sharp increase in DPC concentrations indicating the potential saturation of the detoxification pathways (Casanova *et al.*, cited in OECD, 2002).

A significant correlation between the formation of tumour cells and cell proliferation was reported in rat nasal cavities after inhalation exposures of 10 and 15 ppm (12 and 19 mg m^{-3}). Cell proliferation was not observed when exposure concentrations were equal to or less than 6 ppm (7.5 mg m^{-3}) (Monticello *et al.*, cited in ATSDR, 1999). Repeated and prolonged damage to the nasal epithelium associated with chronic exposure to formaldehyde at irritating concentrations appears to be required for the subsequent development of squamous cell carcinoma in rats. (Bhalla *et al.*, Monteiro-Riviere and Popp, Wilmer *et al.*, Chang *et al.*, Feron *et al.*, Rusch *et al.*, Woutersen *et al.*, Woutersen *et al.*, cited in ATSDR, 1999).

Thus formaldehyde can be described as a whole carcinogen; it is weakly genotoxic, can directly impact DNA (forming DPCs), promotes cell proliferation and progression (damages cells by direct irritation) (IARC, 2004; WHO, 2002; OECD, 2002; ATSDR, 1999). However, formaldehyde's carcinogenic properties appear to be due to its cytotoxic properties (EC, 2001; OECD, 2002).

The U.S. EPA classifies formaldehyde as a *B1: probable human carcinogen* (IRIS, 1991). An IARC Working Group in 2003 (IARC, 2004) re-classified formaldehyde as *carcinogenic to humans (Group 1)*; previous assessment classified formaldehyde as *probably carcinogenic to humans (Group 2A)* (IARC, 1995).

The U.S. EPA reports sufficient evidence of data for carcinogenicity in animals and classifies formaldehyde as a *B1 - probable human carcinogen* (1991).

4.3 Acute and Subacute Effects

Acute effects usually occur rapidly as a result of short-term exposures that are of short duration – generally for exposures less than 24 hours. Subacute effects usually occur as a result of exposures that are of an intermediate duration – generally for exposures lasting a few days to no greater than one month (Eaton and Klaassen, 1996).

4.3.1 Acute and Subacute Human Effects

Some of the human inhalation exposure data was collected after occupational exposures. There are a number of limitations to be considered when using data from people exposed in the work place: i) the person exposed generally is a healthy, young to middle aged, male adult; ii) concurrent exposures to other chemicals are very likely; and, iii) the exposure concentrations are often difficult to define.

Table 6 lists some examples of the lowest and highest NOAELs (No Observable Adverse Effect Level) and LOAELs (Lowest Observable Adverse Effect Level) reported in the literature from acute human exposures. No reports of subacute human exposures were identified.

4.3.1.1 Respiratory and Ocular Effects

ATSDR (1999) listed over 15 studies of acute human exposures which reported primarily irritation of the eyes, nose, and throat; a selection of these studies is provided in Table 6. The tissues predominantly affected by acute formaldehyde inhalation are the tissues of the upper respiratory tract. Exposures to concentrations of 0.2 to 3 ppm (0.25 to 3.7 mg m^{-3}) were mildly to moderately irritating to the nose and throat. Effects did not seem to vary with respect to the health status of the individual (healthy or asthmatic). Eye irritation was also reported after acute airborne exposure (0.2 – 0.5 ppm; 0.25 – 0.62 mg m^{-3}) (ATSDR, 1999; EC, 2001; OECD, 2002). Eye irritation appears to be a more sensitive end point than respiratory irritation (OECD, 2002).

Many controlled exposure studies did not report significant changes in pulmonary function variables (forced vital capacity, forced expiratory volume, peak expiratory flow rate, and forced expiratory flow rate) in health and asthmatic volunteers (Andersen and Molhave; Day *et al.*; Gorski *et al.*, Harving *et al.*, Krakowiak *et al.*, Kulle *et al.*, Kulle, Reid and Figas, Schachter *et*

Table 2 NOAELs and LOAELs for Acute Formaldehyde Inhalation (Human)

Effects Reported[a]	Exposure Period	Air Concentration [b] ppm (mg m^{-3})	Reference
Systemic: Respiratory Tract			
Nose and throat irritation (healthy subjects). NOAEL.	3 hr	1 (1.2)	Kulle *et al.*; Kulle, cited in ATSDR, 1999.
Nasal irritation (health subjects). Less serious LOAEL.	4 hr	0.2 (0.25)	Andersen and Molhave, cited in ATSDR, 1999.
Respiratory effects (decreased PEFR>15%) (purported asthmatics). NOAEL.	30 min	1 (1.2)	Nordman *et al.*, cited in ATSDR, 1999.
Nose/throat irritation, some changes in pulmonary variables (FEV_1>10% in 5/38). Less serious LOAEL.	1 hr	3 (3.7)	Green *et al.*, cited in ATSDR, 1999.
Nasal irritation (two subject groups: asthmatic and healthy). Less serious LOAEL.	40 min	2 (2.5)	Witek *et al.*; Witek *et al.*, cited in ATSDR, 1999.
Nose irritation (health subjects). Less serious LOAEL.	3 hr	1.88 (2.35)	Akbar-Khanzahdeh and Mlynek, cited in ATSDR, 1999.
Systemic: Other			
Eye irritation (health subjects). NOAEL.	3 hr	0.5 (0.6)	Kulle *et al.*, Kulle, cited in ATSDR, 1999.
Eye irritation (health subjects). Less serious LOAEL.	3 hr	1.88 (2.35)	Akbar-Khanzahdeh and Mlynek, cited in ATSDR, 1999.
Eye irritation (two subject groups: asthmatic and healthy). Less serious LOAEL.	40 min	2 (2.5)	Witek *et al.*, Witek *et al.*, cited in ATSDR, 1999.
Eye irritation (health subjects). Less serious LOAEL.	4 hr	0.2 (0.25)	Andersen and Molhave, cited in ATSDR, 1999.
Increased eosinophils and protein in nasal lavage fluid (non-pre-exposed and asthmatic subjects). Less serious LOAEL.	2 hr	0.4 (0.5)	Krakowiak *et al.*, cited in ATSDR, 1999.
Immunological/Lymphoreticular:			
NOAEL.	3 hr	1.0 (1.2)	Pross *et al.*, cited in ATSDR, 1999.
Neurological:			
Decreased performance on short-term memory tests. Less serious LOAEL.	5.5 hr	0.12 (0.15)	Bach *et al.* cited in ATSDR, 1999.

[a] NOAEL, Less serious LOAEL, and Serious LOAEL as identified by ATSDR (1999).

[b] When both units of concentration were not provided in the literature, the following conversion factor and assumptions were used: mg m^{-3} x 24.45/MW =ppm; MW=30.03, air at 25°C and 101.3 kPa (760mmHg) (Plog *et al.*, 1996).

PEFR – Peak Expiratory Flow Rate; FEV_1 - Forced Expiratory Volume in 1.0 second

al.; Witek *et al.*, cited in ATSDR, 1999). A few studies observed some change in pulmonary function with low exposures (1 to 3 ppm, 1.2 to 3.7 mg m^{-3}), but the effects were subtle and infrequent; these effects appeared to be independent of the respiratory health of the subjects (healthy/asthmatic) (Green *et al.*; Nordman *et al.*; Sauder *et al.*, cited in ATSDR, 1999). A number of the studies observed decreased adverse effects with continued exposure (Bender *et al.*; Day *et al.*; Green *et al.*; Weber-Tschopp *et al.*, cited in ATSDR, 1999).

Eye, nose, and throat irritation were reported after short term (4 hours or less) inhalation of formaldehyde (0.2 to 3.6 mg m^{-3}; 0.25 to 4.5 ppm) citing similar studies to ATSDR (1999) (Andersen and Mølhave; Saunder *et al.*; Saunder *et al.*; Schachter *et al.*; Green *et al.*; Green *et al.*; Witek *et al.*; Kulle; Pazdrack *et al.*, cited in EC, 2001). Individual study details were not provided (EC, 2001). Brief exposures (up to 3 hours) to concentrations less than 3.6 mg m^{-3} (2.9 ppm) did not result in significant clinical respiratory effects in both healthy and asthmatic individuals (Day *et al.*; Sauder *et al.*; Schacter *et al.*; Green *et al.*; Witek *et al.*; Harving *et al.*, cited in EC, 2001).

Similar variations in pulmonary function tests were observed in studies of occupational exposures (Akbar-Khanzadeh *et al.*, Akbar-Khanzadeh and Mlynek, Alexandersson and Hedenstierna, Brachen *et al.*, Holness and Nethercott, Horvath *et al.*, Kilburn *et al.*, Malaka and Kodama, cited in ATSDR, 1999).

No studies of higher, acute exposure concentrations were identified in humans (EC, 2001; OECS, 2002; ATSDR, 1999).

4.3.1.2 Other Effects

A single study of potential neurological effects after acute inhalation reported decreased performances (distractibility, short-term memory, capability to understand and perform certain tasks) in exposed subjects with increased formaldehyde exposure concentrations. Four exposure concentrations were used (0.0, 0.12, 0.32, and 0.98 ppm; 0.0, 0.15, 0.4 and 1.2 mg m^{-3}); no respiratory effects were observed (Bach *et al.*, cited in ATSDR, 1999).

There was no significant change in serum formaldehyde specific IgE antibodies after inhalation challenge (Dykewicz *et al.*; Grammar *et al.*; Kramps *et al.*, Wantke *et al.*, Wantke *et al.*, cited in ATSDR, 1999).

No reports of deaths due to acute formaldehyde inhalation were identified (ATSDR, 2001; EC, 2001; OECD, 2002).

4.3.2 Acute and Subacute Animal Effects

Table 7 lists some examples of the lowest and highest NOAELs (No Observable Adverse Effect Level) and LOAELs (Lowest Observable Adverse Effect Level) reported in the literature from acute animal exposures. Table 8 lists some examples of the lowest and highest NOAELs (No

Observable Adverse Effect Level) and LOAELs (Lowest Observable Adverse Effect Level) reported in the literature from sub-acute animal exposures.

4.3.2.1 Respiratory Effects

Acute and sub-acute exposures in laboratory studies of mice, rats, and monkeys demonstrated that the tissues primarily affected by formaldehyde inhalation are in the upper respiratory tract. The specific location and the cells affected are species dependant due to differences in respiratory physiology (ATSDR, 1999).

In rats, acute exposures greater than 2 to 6 ppm (2.5 to 7.5 mg m^{-3}) generally resulted in epithelial damage (histological legions and increased rates of cell proliferation) in the nasal cavity (Bhalla *et al.*; Cassee and Feron; Monteiro-Riviere and Popp; Monticello *et al.*; Morgan *et al.*; Wilmer *et al.*, cited in ATSDR, 1999). Sub-acute exposures (6 ppm; 7.5 mg m^{-3}) in monkeys produced significant epithelial lesions in the upper respiratory tract, and to a lesser extent in tracheal and major bronchial tissues (Monticello *et al.*, cited in ATSDR, 1991).

Acute formaldehyde exposure affected lower airway resistance and hyper-reactivity of the lungs in guinea pigs (Amdur; Swiecichowski *et al.*, cited in ATSDR, 1999).

Epithelial damage to the upper respiratory tissues in rats appears to occur at a lower exposure concentration than mice; however, mice appear to be more sensitive to formaldehyde-induced sensory irritation (characterized by reduced respiratory rate and tidal volume) (Chang *et al.*; Kane and Alarie, cited in ASDR, 1999). The latter effect may explain why mice are less sensitive to formaldehyde-induced tissue damage than rats; a reduced respiration rate, and thus reduced penetration of formaldehyde into the respiratory tract (ATSDR, 1999). As discussed in Section 4.1 formaldehyde is rapidly absorbed and metabolized, therefore is most likely to affect tissues with which there is direct contact.

Acute damage to the lung tissues in rats occurred only at concentrations higher than those associated with damage to the upper respiratory tissues. No damage to lung tissues was reported after acute exposures to 10 ppm (12.5 mg m^{-3}); however, exposure to much higher concentrations (150 ppm up to 295 ppm; 188 to 369 mg m^{-3}) produced histological changes in the nasal turbinates, the trachea, and the lung. Exposure to the higher concentrations (128 or 295 ppm; 160 or 369 mg m^{-3}) induced bloody nasal discharge and pulmonary oedema (Dinsdale *et al.*, Kamata *et al.*, cited in ATSDR, 1999).

Table 3 NOAELs and LOAELs for Acute Formaldehyde Inhalation (Experimental Animals)

Effects Reported[a]	Exposure Period	Air Concentration[b] ppm (mg m^{-3})	Species	Reference
Death:				
LC_{50}.	30 min	816 (984)	Rat	Skog, cited in OECD, 2002.
LC_{50}.	4 hr	480 (578)	Rat	Nagorny *et al.*, Cited in OECD, 2002.
LC_{50}s	"acute"	394 - 786 (493 – 984)	Rodents	WHO cited in EC 2001.
Dyspnea, vomiting, hypersalivation, muscle spasms, convulsions, and death.	"acute"	96 (>120)	Not specified.	Skog; WHO, cited in OECD, 2002. WHO cited in EC 2001.
Systemic:				
Alterations in mucociliary clearance and nasal histopathological changes.	"acute"	2.1 (≥2.6)	Rat	Monteiro-Riviere and Popp; Morgan *et al*; Bhalla *et al.*, cited in EC 2001.
Hypertrophy in nasal passages. Serious LOAEL.	6 hr, 1,2,or 9 d	6 (7.5)	Rat	Monteiro-Riviere and Popp, cited in ATSDR, 1999.
Ciliary destruction and cell separation in naso- and maxillo-turbinates, cellular swelling throughout turbinates, mucous releasing goblet cells in naso-turbinates. Serious LOAEL.	4 hr	10 (12.5)	Rat	Bhalla *et al.*, cited in OECD, 2002 and ATSDR, 1999.
Nasal epithelial cell necrosis; neutrophil infiltrations; epithelial hyperplasia; squamous metaplasia; increased cell proliferation. NOAEL.	6 hr, 1,2,or 9 d	2 (2.4)	Rat	Monticello *et al.*, cited in ATSDR, 1999 and EC 2001.
Nasal epithelial cell necrosis; neutrophil infiltrations; epithelial hyperplasia; squamous metaplasia; increased cell proliferation. Serious LOAEL.	6 hr, 1,2,or 9 d	6.0 (7.4)	Rat	Monticello *et al.*, cited in ATSDR, 1999 and EC 2001.
Bloody nasal discharge; pulmonary edema. Serious LOAEL.	6 hr	128 (160)	Rat	Kamata *et al.*, cited in ATSDR, 1999.
Respiratory effect (RD_{50}). Less serious LOAEL.	10 min.	4.9 (6.1)	Mice	Chang *et al.*, cited in ATSDR, 1999.
Increased nasal respiratory epithelial cell turnover; mild to serious rhinitis and focal degeneration of the respiratory epithelium; congestion of the olifactory blood vessels, focal erosion and ulceration; hyperplasia. Serious LOAEL.	6 hr/d, 1 or 5 d	15 (19)	Mice	Chang *et al.*, cited in ATSDR, 1999.

Table 7 NOAELs and LOAELs for Acute Formaldehyde Inhalation (Experimental Animals) (continued)

Effects Reported[a]	Exposure Period	Air Concentration[b] ppm (mg m^{-3})	Species	Reference
Increased airway resistance. NOAEL	2 hr	3.4 (4.2)	Male guinea pig	Swiecichowski *et al.*, cited in ATSDR, 1999.
Increased airway resistance. Less serious LOAEL	2 hr	9.4 (12)	Male guinea pig	Swiecichowski *et al.*, cited in ATSDR, 1999.
Increased airway resistance. NOAEL	8 hr	0.1 (0.12)	Guinea pig	Swiecichowski *et al.*, cited in ATSDR, 1999.
Increased airway resistance. Less serious LOAEL	2 hr	0.3 (0.38)	Male guinea pig	Swiecichowski *et al.*, cited in ATSDR, 1999.
Neurological:				
Restlessness. Less serious LOAEL.	10 min – 6hr	15 (19)	Male rat	Morgan *et al.*, cited in ATSDR, 1999.
Decreased motor activity; increased concentrations of 5-hydroxyindoleacetic acid, 3,4-dihydroxyphenyl-acetic acid and dopamine in the hypothalamus. Less serious LOAEL.	3 hr/d, 1-2d	5 (6.2)	Male rat	Boja *et al.*, cited in ATSDR, 1999.

[a] NOAEL, Less serious LOAEL, and Serious LOAEL as identified by ATSDR (1999).
[b] When both units of concentration were not provided in the literature, the following conversion factor and assumptions were used: mg m^{-3} x 24.45/MW =ppm; MW=30.03, air at 25°C and 101.3 kPa (760mmHg) (Plog *et al.*, 1996).

4.3.2.2 Other Effects

Some behavioural effects and changes neurological chemistry (acute in halation exposures) have been reported (Morgan *et al.*; Boja *et al.*, cited in ATSDR, 1999).

Three studies reported some immunological effects (humoral response; bronchial sensitization; increase sensitization to respiratory allergens) with formaldehyde inhalation (Tarkowski and Gorski; Riedel *et al.*, cited in ATSDR, 1999 and EC, 2001. Adams *et al.*, cited in ATSDR, 1999). A significant reduction in maternal body weight was observed in rats; no developmental or reproductive effects were observed unless maternal health was compromised (Saillenfait *et al.*, cited in ATSDR, 1999 and EC, 2001; Martin, cited in EC, 2001).

Table 4 NOAELs and LOAELs for Subacute Formaldehyde Inhalation (Experimental Animals)

Effects Reported[a]	Exposure Period	Air Concentration[b] ppm (mg m^{-3})	Species	Reference
Systemic:				
Increased cell proliferation in nasal cavity. NOAEL.	6 hr/d, 3 d.	2 (2.4)	Rat	Swenberg *et al.*; Swenberg *et al.*, cited in EC 2001.
Histopathological effects in nasal cavity; inhibition of mucociliary clearance. NOAEL	6 hr/d, 5 d/wk, 1, 2, 4, or 14 d	2 (2.4)	Rat	Morgan *et al.*, cited in EC 2001.
Increased cell proliferation in nasal cavity. NOAEL.	6 hr/d, 3 d.	6 (7.2)	Mice	Swenberg *et al.*; Swenberg et al., cited in EC 2001.
Hyperplasia and squamous metaplasia in nasal epithelium, extending to trachea and carina (no effects on the lungs or other internal organs or systems). Serious LOAEL.	6 hr/d, 5 d/wk, 1 or 6 wk	6 (7.2)	Male monkeys	Monticello *et al.*, cited in ATSDR, 1999 and EC, 2001.
Mild lacrimation and conjunctival hyperemia. Less serious LOAEL.	6 hr/d, 5 d	6 (7.2)	Male monkeys	Monticello *et al.*, cited in ATSDR, 1999.
Body weight. NOAEL.	6 hr/d, 5 d/wk, 3 wk	15 (19)	Female mice	Adams *et al.*, cited in ATSDR, 1999.
Immunological/Lymphoreticular:				
NOAEL.	6 hr/d, 5 d/wk, 3 wk	15 (19)	Female mice	Dean *et al.*, cited in ATSDR, 1999.
Increased ability to release reactive oxygen intermediates. Less Serious LOAEL.	6 hr/d, 5 d/wk, 3 wk	15 (19)	Female mice	Adams *et al.*, cited in ATSDR, 1999.
Increased IgE response to inhaled ovalbumin. Less serious LOAEL.	6 hr/d, 10 d	1.6 (2.0)	Mice	Tarkowski and Gorski, 1995 cited in ATSDR, 1999 and EC, 2001.
Allergic response to ovalbumin. Less serious LOAEL.	8 hr/d, 5 d	0.25 (0.31)	Guinea pig	Riedel *et al.*, 1996 cited in ATSDR, 1999 and EC, 2001.

Table 8 NOAELs and LOAELs for Subacute Formaldehyde Inhalation (Experimental Animals) (continued)

Effects Reported[a]	Exposure Period	Air Concentration [b] ppm (mg m^{-3})	Species	Reference
Reproductive and Developmental:				
Reproductive. NOAEL.	6 hr/d, 15 d, gd[c] 60-20	40 (50)	Female rats	Saillenfait *et al.*, cited in ATSDR, 1999.
Developmental (decreased foetal weight). NOAEL.	6 hr/d, 15 d, gd[c] 60-20	10 (12.5)	Female rats	Saillenfait *et al.*, cited in ATSDR, 1999.
Body weight (maternal). NOAEL.	6 hr/d, 15 d, gd[c] 60-20	20 (25)	Female rats	Saillenfait *et al.*, cited in ATSDR, 1999.
Body weight (maternal). NOAEL.	6 hr/d, 10 d, gd[c] 6-15	5 (6)	Female rats	Martin, cited in EC, 2001.
Body weight (maternal). Serious LOAEL.	6 hr/d, 15 d, gd[c] 60-20	40 (50)	Female rats	Saillenfait *et al.*, cited in ATSDR, 1999.
Developmental (5% decrease in foetal weight). Less Serious LOAEL.	6 hr/d, 15 d, gd[c] 60-20	20 (25)	Female rats	Saillenfait *et al.*, cited in ATSDR, 1999.
Developmental (21 % decrease in foetal weight). Serious NOAEL.	6 hr/d, 15 d, gd[c] 60-20	40 (50)	Female rats	Saillenfait *et al.*, cited in ATSDR, 1999.

[a] NOAEL, Less serious LOAEL, and Serious LOAEL as identified by ATSDR (1999).
[b] When both units of concentration were not provided in the literature, the following conversion factor and assumptions were used: mg m^{-3} x 24.45/MW =ppm; MW=30.03, air at 25°C and 101.3 kPa (760mmHg) (Plog *et al.*, 1996).
[c] gd-gestational day.

4.4 Chronic Effects

4.4.1 Chronic Human Effects

Subchronic or intermediate exposures are generally one to three months; chronic effects occur as a result of long-term exposures and are of longer duration – generally as repeated exposures for more than 3 months (Eaton and Klaassen, 1996).

The majority of human inhalation exposure data available has been collected after occupational exposures. There are a number of limitations to be considered when using data from people exposed in the work place: i) the person exposed generally is a healthy, young to middle aged, male adult; ii) concurrent exposures to other chemicals are very likely, and; iii) the exposure

concentrations are often difficult to define. Table 9 lists some of the lowest and highest NOAELs and LOAELs reported in the literature.

Table 5 NOAELs and LOAELs for Subchronic and Chronic Formaldehyde Inhalation (Human)

Effects Reported[a]	Exposure Period	Air Concentration [b] ppm (mg m^{-3})	Reference
Systemic: Respiratory Tract			
Furniture factory workers. NOAEL.	7.3 yr (range 1-36 yr)	0.2 (0.25)	Holmstrom *et al.*, cited in ATSDR, 1999.
Plywood factory workers. Increased lesions in nasal epithelium samples. Less Serious LOAEL.	6.8 yr (range 2-19 yr)	0.39 (0.49)	Ballarin *et al.*, cited in ATSDR, 1999.
Male particleboard workers. Increased lesions in nasal epithelium cells (nonciliated cells, metaplasia, mild dysplasia). Less Serious LOAEL.	10.5 yr (range 1-39 yr)	0.49 (0.61)	Edling *et al.*, cited in ATSDR, 1999.
Chemical workers. Increased lesions in nasal epithelium samples (nonciliated cells, metaplasia, mild dysplasia). Less Serious LOAEL.	7.3 yr (range 1-36 yr)	0.24 (0.3)	Holmstrom *et al.*, cited in ATSDR, 1999.
Embalmers. Increased reports of respiratory irritation. Less Serious LOAEL	8.2 yr (range not reported)	0.36 (0.45)	Holness and Nethercott, cited in ATSDR, 1999.
Particleboard workers. Increased reporting of respiratory symptoms. Less Serious LOAEL.	10.3 yrs (range <1-20 yr)	0.69 (0.86)	Horvath *et al.*, cited in ATSDR, 1999.
Systemic: Other			
Male particleboard workers. Running eyes (75%). Less Serious LOAEL.	10.5 yr (range 1-39 yr)	0.49 (0.61)	Edling *et al.*, cited in ATSDR, 1999.
Embalmers. Increased eye irritation. Less Serious LOAEL.	8.2 yr (range not reported)	0.36 (0.45)	Holness and Nethercott, cited in ATSDR, 1999.
Particleboard workers. Increased report of itchy burning/watery eyes. Less Serious LOAEL.	10.3 yrs (range <1-20 yr)	0.69 (0.86)	Horvath *et al.*, cited in ATSDR, 1999.
Embalmers. Increase in past skin problems and contact dermatitis. Less Serious LOAEL.	8.2 yr (range not reported)	0.36 (0.45)	Holness and Nethercott, cited in ATSDR, 1999.

[a] NOAEL, Less serious LOAEL, and Serious LOAEL as identified by ATSDR (1999).

[b] When both units of concentration were not provided in the literature, the following conversion factor and assumptions were used: mg m^{-3} x 24.45/MW =ppm; MW=30.03, air at 25°C and 101.3 kPa (760mmHg) (Plog *et al.*, 1996).

Table 6 Examples of Case-Controlled Studies of Formaldehyde Inhalation (Human) (EC, 2001 and WHO, 2002)

Cancer[a]	Exposure	Risk Measure (95% CI)	Reference (comments)[b]
Oropharynx or hypopharynx. SEER population based – Washington state.	>10 yr occupational exposure Occupational score of >20	OR = 1.3 (0.7-2.5) OR = 1.5 (0.7-3.0)	Vaughan *et al.*, cited in EC, 2001, WHO, 2002. (IARC Working Group noted: different proportions of interviews conducted with next-of-kin cases; and, controls may have affected the odds ratio)
Nasopharynx. SEER population based – Washington state.	Exposure score >20	OR = 2.1 (0.6-7.8)	Vaughan *et al.*, cited in EC, 2001, WHO, 2002. (IARC Working Group noted: different proportions of interviews conducted with next-of-kin cases; and, controls may have affected the odds ratio)
Nasopharynx. SEER population based – Washington state.	Residential score of >10 yr Residential score of <10 yr	OR = 5.5 (1.6-19.4) OR = 2.1 (0.7-6.6)	Vaughan *et al.*, cited in EC, 2001, WHO, 2002. (IARC Working Group noted: different proportions of interviews conducted with next-of-kin cases; and, controls may have affected the odds ratio)
Nasal Squamous cell carcinoma Hospital based – Netherlands	Occupational exposure assessment A Occupational Assessment B	OR = 3.0 (1.3-6.4)[b] OR = 1.9 (1.0-3.6)[b]	Hayes *et al.*, cited in EC, 2001, WHO, 2002. (IARC Working Group noted: a greater proportion of cases were dead; variable numbers of next-of-kin were interviewed, 10% of controls but none of cases, by telephone; although different, results for assessments A & B were both positive)
Squamous cell carcinoma of nasal cavity/paranasal sinus Danish Cancer Registry	Occupational exposure without exposure to wood dust.	OR = 2.0 (0.7-5.9)	Olsen and Asnaes, cited in EC, 2001, WHO, 2002. (IARC Working Group noted: possibly incomplete adjustment for confounding wood dust for adenocarcinoma (felt that squamous cell carcinoma less likely to be affected, since no clear association with wood dust); small number of cases).
Nasopharynx. Connecticut Tumour Registry	Highest potential exposure category Highest potential exposure category and dying at 68+ yr	OR = 2.3 (0.9-6.0) OR = 4.0 (1.3-12)	Roush *et al.*, cited in EC, 2001, WHO, 2002.
Oral/oropharynx Population based – Turin, Italy	"Any" occupational exposure "Probable or definite" occupational exposure	OR = 1.6 (0.9-2.8) OR = 1.8 (0.6-5.5)	Merletti *et al.*, cited in EC, 2001, WHO, 2002. (small number of cases with "definite" exposure)[d]

Table 10 Examples of Case-Controlled Studies of Formaldehyde Inhalation (Human) (EC, 2001 and WHO, 2002) (continued)

Cancer[a]	Exposure	Risk Measure (95% CI)	Reference (comments)[b]
Larynx SEER population based – Washington State.	"High" occupational exposure Occupational Exposure >10 yr Occupational exposure score >20 yr	OR = 2.0 (0.2-19.5) OR = 1.3 (0.6-3.1) OR = 1.3 (0.5-3.3)	Wortley *et al.*, cited in EC, 2001, WHO, 2002.
Nasal cavity/paranasal sinus (adenocarcinoma). Population based - France	"Any" exposure (no wood dust). "Any" exposure (medium to high wood dust). "No" exposure (medium to high wood dust)	OR = 8.1 (0.9-72.9) OR = 692 (91.9-5210) OR = 130 (14.1-1191)	Luce *et al.*, cited in EC, 2001, WHO, 2002. (IARC Working Group noted: possible residual confounding by exposure to wood dust).
Nasopharynx. Hospital based – Philippines	<15yr exposure. >25yr exposure. <25 years of age at first exposure.	OR = 2.7 (1.1-6.6) OR = 2.9 (1.1-7.6) OR = 2.7 (1.1-6.6)	West *et al.*, cited in EC, 2001, WHO, 2002. (IARC Working Group noted: no control for the presence of Epstein-Barr viral antibodies (for which a previous strong association with nasopharyngeal cancer was observed).
Lung. Nested – cohort of chemical workers – Texas.	Likely occupational exposures.	OR = 0.62 (0.29-1.36)	Bond *et al.*, cited in EC, 2001, WHO, 2002..
Lung. Lung (adenocarcinoma) Population based – Montréal, Quebec	"Long-high" occupational exposure/(cancer controls/population controls) "Long-high" occupational exposure/(cancer controls/population controls)	OR = 1.5 (0.8-2.8)/ OR = 1.0 (0.4-2.4) OR = 2.3 (0.9-6.0)/ OR = (2.2 (0.7-7.6)	Gérin *et al.*, cited in EC, 2001, WHO, 2002..
Respiratory Cancer. Nested – cohort of Finish woodworkers.	Cumulative exposures of >3.6 mg m^{-3}-months, without a minimum 10-yr induction period. Cumulative exposures of >3.6 mg m^{-3}-months, with a minimum 10-yr induction period. Exposure to formaldehyde in wood dust.	OR = 0.69 (0.21-2.24)[b] OR = 0.89 (0.26-3.0)[b] OR = 1.19 (0.31-4.56)[b]	Partanen *et al.*, cited in EC, 2001, WHO, 2002. (IARC Working Group noted: were too few cancers at sites other than lung for meaningful analysis).

Table 10 Examples of Case-Controlled Studies of Formaldehyde Inhalation (Human) (EC, 2001 and WHO, 2002) (continued)

Cancer[a]	Exposure	Risk Measure (95% CI)	Reference (comments)[b]
Lung. Population based – Missouri	Potentially exposed non-smokers.	OR = 0.9 (0.2-3.3)	Brownson *et al.*, cited in EC, 2001, WHO, 2002.
Lung. Nested – cohort of US automotive foundry workers.	Occupational exposure with latency period of: 0 yr 10 yr 15 yr 20 yr	 OR = 1.31 (0.93-1.85) OR = 1.04 (0.71-1.52) OR = 0.98 (0.65-1.47) OR = 0.99 (061-162)	Andejlkovich *et al.*, cited in EC, 2001, WHO, 2002.
Multiple myelenoma Incident cases in follow-up of cancer prevention study in the U.S.	Probably exposed	OR = 1.8 (0.6-5.7)	Boffetta *et al.*, cited in EC, 2001, WHO, 2002.
Multiple mylenoma Danish Cancer Registry	Males with probable occupational exposure Females with probable occupational exposure	OR = 1.1 (0.7-1.6) OR = 1.6 (0.4-5.3)	Heineman *et al.*; Pottern *et al.*, cited in EC, 2001, WHO, 2002.
Non-Hodgkin's lymphoma Iowa State Health Registry	Potential "lower intensity" of exposure Potential "higher intensity" of exposure	OR = 1.2 (0.9-1.7) OR = 1.3 (0.5-3.8)	Blair *et al.*, cited in EC, 2001, WHO, 2002.
Ocular melanoma Cases diagnosed or treated at UCSF Ocular Oncology Unit	"Ever" exposed to formaldehyde	OR = 2.9 (1.2-7.0)	Holly *et al.*, cited in EC, 2001, WHO, 2002.

[a] SEER – Surveillance, Epidemiology, and End Results program of the National Cancer Institute; UCSF – University of California at San Francisco.
[b] Data in parenthesis = 90% confidence interval.
[c] Interpretive comments as reported in: EC, 2001 and CICAD, 2002.
[d] Interpretive comment as reported in EC, 2001.
CI – Confidence Interval.
OR – Odds Ratio

Table 7 Examples of Cohort Studies of Formaldehyde Inhalation (Human) (EC, 2001 and CICAD, 2002)

Cancer	Cohort Exposed	Risk Measure (95% CI)	Reference (comments)[a]
Brain	Male anatomists	SMR = 2.7 (1.3-5.0): 10	Stroup *et al.*, cited in EC, 2001, WHO, 2002.
Leukemia		SMR = 1.5 (0.7-2.7): 10	
"Other lymphatic tissues"		SMR = 2.0 (0.7-4.4): 6	(Likely exposure to other substances; no
Nasal cavity and sinus		SMR = 0 (0.7-7.2): 0	qualitative data on exposure)
Larynx		SMR = 0.3 (0-2): 1	
Lung		SMR = 0.3 (0.1-0.5): 12	
Multiple myeloma	Male abrasive production workers	SIR = 4 (0.5-14): 2	Edling *et al.*, cited in EC, 2001, WHO, 2002.
Lymphoma		SIR = 2 (0.2-7.2): 2	
Pancreas		SIR = 1.8 (0.2-6.6): 2	(Increases based on only two cases)
Lung		SIR = 0.57 (0.1-2.1): 2	
Buccal cavity	Garment factory workers	SMR = 343 (118-786)[b]: 4	Stayner *et al.*, cited in EC, 2001, WHO, 2002.
Connective tissue		SMR = 364 (123-825)[b]: 4	
Trachea, bronchus, lung		SMR = 114 (86-149)[b]: 39	
Pharynx		SMR = 111 (20-359)[b]: 2	
Alimentary tract	Resin manufacturing workers	SMR = 134 (p>0.05): 11	Bertazzi *et al.*, cited in EC, 2001, WHO, 2002.
Stomach		SMR = 164 (p>0.05): 5	
Liver		SMR = 244 (p. >0.05): 2	(Small cohort exposed primarily to low
Lung		SMR = 69: 6	concentrations: few deaths during observation period)
Buccal cavity and pharynx	Male Pathologists	SMR = 0.52 (0.28-0.89): 13	Matanoski, cited in EC, 2001, WHO, 2002.
Respiratory system		SMR = 0.56 (0.44-0.77): 77	
Hypopharynx		SMR = 4.7 (0.97-): 3	
Pancreas		SMR = 1.4 (1.04-1.88): 47	
Leukemia		SMR = 1.68 (1.14-2.38): 31	
Buccal cavity and pharynx	Male mortuary workers	PMR = 120 (81-171): 30	Hayes *et al.*, cited in EC, 2001, WHO, 2002.
Nasopharynx		PMR = 216 (59-544): 4	
Lymphatic and haematopoietic		PMR = 139 (115-167): 115	
Colon		PMR = 127 (104-153): 111	
Trachea, bronchus, lung		PMR = 94.9: 308	

Table 11 Examples of Cohort Studies of Formaldehyde Inhalation (Human) (EC, 2001 and CICAD, 2002) (continued)

Cancer	Cohort Exposed	Risk Measure (95% CI)	Reference (comments)[a]
Lung	Male chemicals workers employed before 1965.	SMR = 123 (110-136): 348	Gardner at al., cited in EC, 2001, WHO, 2002.
Buccal cavity		SMR = 137 (28-141): 3	
Pharynx		SMR = 147 (59-303): 7	(35% of cohort exposed to > 2 ppm (2 mg m^{-3})
Lung	Workers exposed to >2.4 mg m^{-3} (1.9 ppm) at one specific plant	SMR = 126 (107-147): 165	
Nasal cavity	Male industrial workers	SPIR = 2.3 (1.3-4.0): 13	Hansen and Olsen, cited in EC, 2001, WHO, 2002.
Nasopharynx		SPIR = 1.3 (0.3-3.2): 4	
Lung		SPIR = 1.0 (0.9-1.1): 410	
Larynx		SPIR = 0.9 (0.6-1.2): 32	
Oral Cavity and pharynx		SPIR = 1.1 (0.7-1.7): 23	
Nasal cavity	Male industrial workers exposed above baseline levels	SPIR = 3.0 (1.4-5.7): 9	
Buccal cavity and pharynx	Male automotive foundry worker	SMR = 131 (48-266): 6	Andjelkovich *et al.*, cited in EC, 2001, WHO, 2002.
Trachea, bronchus, lung		SMR = 120 (89-159): 51	(25% of cohort exposed to >1.5 ppm (1.8 mg m^{-3}))
Nasopharynx	White male industrial workers exposed to >0.1 ppm (0.12 mg m^{-3})	SMR = 2.7 ($p<0.05$): 6	Blair *et al.*, cited in EC, 2001, WHO, 2002.
			(4% of cohort exposed to >2 ppm (2.4 mg m^{-3}))
	White male industrial workers with cumulative exposures of:		
	0 ppm-years	SMR = 503: 1	
	<0.5 ppm years	SMR = 271 ($p>0.05$): 2	
	0.51-5.5 ppm years	SMR = 256 ($p>0.05$): 2	
	>5.5 ppm-years	SMR =433 ($p>0.05$): 2	
Nasopharynx	White male industrial workers:		Collins *et al.* cited in EC, 2001, WHO, 2002.
	Exposed for <1 yr	SMR = 517 ($p<0.05$): 3	
	Exposed for >1 yr	SMR = 218 ($p<0.05$): 3	
	Exposed at one plant with particulates	SMR = 1031 ($p<0.01$): 4	
Nasopharynx	White male workers, hired between 1947 – 1956, employed at one specific plant for:		Marsh *et al.*, cited in EC, 2001, WHO, 2002.
	<1 yr	SMR = 768 ($p>0.05$): 2	
	>1 yr	SMR = 1049 ($p>0.05$): 2	

Table 11 Examples of Cohort Studies of Formaldehyde Inhalation (Human) (EC, 2001 and CICAD, 2002) (continued)

Cancer	Cohort Exposed	Risk Measure (95% CI)	Reference (comments)[a]
Lung	White male industrial workers exposed to >0.1 ppm (0.12 mg m^{-3})	SMR = 111 (96-127):210	Blair *et al.*, cited in EC, 2001, WHO, 2002.
	White male industrial workers with >20 years since first exposure	SMR = 132 ($p<0.05$): 151	(4% of cohort exposed to >2 ppm (2.4 mg m^{-3}))
	White male industrial workers with cumulative exposures of:		
	0 ppm-years	SMR = 68 (37-113): 14	
	<0.5 ppm-years	SMR = 122 (98-150): 88	
	0.51-5.5 ppm-years	SMR = 100 (80-124):86	
	>5.5 ppm-years	SMR = 111 (85-143):62	
Lung	Wage-earning white males in industrial cohort exposed to formaldehyde and other substances.	SMR = 1.4 ($p<0.05$): 124	Blair *et al.*, cited in EC, 2001, WHO, 2002.
	Wage-earning white males in industrial cohort exposed to formaldehyde.	SMR = (1.0 ($p>0.05$): 88	
Lung	Subjects in industrial cohort less than 65 years of age with cumulative exposures of:		Sterling and Weinkam, cited in EC, 2001, WHO, 2002.
	<0.1 ppm-years	RR = 1.0	
	0.1-0.5 ppm-years	RR = 1.47 (1.03-2.12)[b]	
	0.5-2.0 ppm-years	RR = 1.47 (1.03-2.12)[b]	
	>2.0 ppm-years	RR = 1.08 (0.67-1.70)[b]	
	Males in industrial cohorts less than 65 years of age with cumulative exposures of:		
	<0.1 ppm-years	RR = 1.0	
	0.1-0.5 ppm-years	RR = 1.50 (1.03-2.19)[b]	
	0.5-2.0 ppm-years	RR = 1.18 (0.73-1.90)[b]	
	>2.0 ppm-years	RR = 1.94 (1.13-3.34)[b]	

Table 11 Examples of Cohort Studies of Formaldehyde Inhalation (Human) (EC, 2001 and CICAD, 2002) (continued)

Cancer	Cohort Exposed	Risk Measure (95% CI)	Reference (comments)[a]
Lung	White wage-earning males in industrial cohort with >2 ppm-years of cumulative exposures and exposure durations of:		Blair and Stewart, cited in EC, 2001, WHO, 2002.
	<1 yr	(no observed deaths)	
	1 - <5yr	SMR = 1.1 (p>0.05): 9	
	5 - <10yr	SMR = 2.8 (p<0.05): 17	
	>10 yr	SMR = 1.0 (p<0.05): 10	
Lung	White male workers employed at one specific plant for:		Marsh *et al.*, cited in EC, 2001, WHO, 2002.
	<1 yr	SMR = 134 (p<0.05): 63	(25% exposed to >0.7 ppm (0.9 mg m^{-3}))
	>1 yr	SMR = 119 (p>0.05): 50	
Lung	White males in industrial cohort with cumulative exposure of:		Callas *et al.*, cited in EC, 2001, WHO, 2002.
	0 ppm-years	RR = 1.00	
	0.05-0.5 ppm-years	RR = 1.46 (0.81-2.61)	
	0.51-5.5 ppm-years	RR = 1.27 (0.72-2.26)	
	>5.5 ppm-years	RR = 1.38 (0.77-2.48)	

[a] Interpretive comments as reported in: EC, 2001 and CICAD, 2002.
[b] Data in parenthesis = 90% confidence interval.
CI – Confidence Interval.
RR – Relative Risk
SMR – Standardized Mortality Ratio
SPIR – Standardized Proportionate Incidence Ratio
PMR – Proportionate Mortality Ratio
SIR – Standardized Incidence Ratio

4.4.1.1 Respiratory Effects

Repeated formaldehyde inhalation (occupational and residential) has been demonstrated to be irritating to the upper respiratory tract (Boyson *et al.*; Edling *et al.*; Garry *et al.*; Holmstrom *et al.*; Holness and Nethercott; Horvath *et al.*; Richie and Lehnen, cited in ATSDR, 1999); however, there was only limited evidence of adverse effects on pulmonary function (Alexanddersson and Hedenstierna; Bracken *et al.*, Holness and Nethercott; Horvath *et al.*, Khamgaonkar and Fulare; Kriebel *et al.*, Krzyzanowski *et al.*, Malaka and Kodama, cited in ATSDR, 1999). Although discussed in the text, not all study details were provided in ATSDR (1999).

Richie and Lehnen (cited in ATSDR, 1999) conducted a survey of persons living in conventional and mobile homes. Reports of eye, nose and throat irritation, headaches, and skin rash were compared to measured home formaldehyde concentrations (low: <0.1 ppm (<0.12 mg m^{-3}); medium: 0.1 – 0.3 ppm (0.12 - 0.37 mg m^{-3}); and, high: >0.3 ppm (0.37 mg m^{-3})). A greater percentage of adverse effects were reported in houses in the medium and high category. However, it is important to note that people included in the study were identified based on previous reports of adverse health effects.

A study of workers in funeral homes and embalmers identified a greater number of respiratory complaints (chronic bronchitis, shortness of breath, and nasal irritation) and eye irritation compared to control subjects after chronic exposures of 0.08-0.81 ppm (0.1-1.01 mg m^{-3}) (Holness and Nethercott, cited in ATSDR, 1999). Workers exposed to formaldehyde (estimated 0.17 – 2.93 ppm; 0.21-3.67 mg m^{-3}) in a particleboard and moulded plastics plant also identified significant increases in respiratory complaints. Workers were also exposed to nuisance particles such as softwood dust (Horvath *et al.*, cited in ATSDR, 1999).

A number of occupational studies examined histological evidence of damage to the nasal tissues of chronically exposed workers (Ballarin *et al.*, Boysen *et al.*, Edling *et al.*, Holmstrom *et al.*, cited in ATSDR, 1999). Significant histological damage was reported in the nasal mucosa of workers:

- in a particleboard processing plant and a laminate plant (exposed to estimated formaldehyde concentrations of 0.08 – 0.9 ppm formaldehyde (0.1-1.1 mg m^{-3}) and wood dust) (Edling et al., cited in ATSDR, 1999);

- in a chemical plant that produced formaldehyde and formaldehyde resins (estimated 0.04–0.4 ppm formaldehyde; 0.05-0.5 mg m^{-3}), but not reported in furniture factory workers (estimated 0.16–0.4 ppm formaldehyde; 0.2-0.5 mg m^{-3}) (furniture workers were exposed to formaldehyde from particle board and glue components and were also exposed to wood dust particulate; severe epithelial changes were reported in two workers) (Holmstom *et al.*, cited in ATSDR, 1999);

- in a chemical plant that produced formaldehyde and formaldehyde resins (estimated 0.5-2.0 ppm formaldehyde; 0.6-2.5 mg m^{-3}) (Boysen *et al.*, cited in ATSDR, 1999); and,

- in a plywood factory (estimated 0.21–0.6 ppm formaldehyde; 0.26-0.75 mg m^{-3}) (workers were exposed to urea formaldehyde glue and wood dust) (Ballarin *et al.*, cited in ATSDR,1999).

Occupational studies demonstrating no significant changes in pulmonary function (e.g., FVC, FEV_1, $FEFR_{25-75}$) included: laboratory workers (estimated 0.106 – 0.259 ppm formaldehyde; 0.132-0.324 mg m^{-3})(Bracken *et al.*, cited in ATSDR, 1999); workers from a particleboard factory (estimated 0.17 – 2.93 ppm formaldehyde; 0.21-3.67 mg m^{-3}) (Horvath *et al.*, cited in ATSDR, 1999); and embalmers (estimated 0.08 – 0.81 ppm formaldehyde; 0.1-1.01 mg m^{-3}) (Holness and Nethercott, cited in ATSDR, 1999) (Boysen *et al.*, cited in ATSDR, 1999). Other studies reporting minimal changes in pulmonary function included:

- workers in a plywood factory (estimated 0.22 – 3.48 ppm formaldehyde; 0.28-4.36 mg m^{-3}) Malaka and Kodama, cited in ATSDR, 1999);

- woodworkers (estimated 0.3 and 0.4 ppm formaldehyde; 0.37 and 0.5 mg m^{-3}) (Alexandersson and Hedenstierna, cited in ATSDR, 1999);

- workers applying lacquer (estimated 0.2 – 2.1 ppm formaldehyde; 0.25 – 2.6 mg m^{-3}) (Alexandersson and Hedenstierna, cited in ATSDR, 1999); and,

- anatomy and histopathology workers (estimated 0.036 – 2.27 ppm formaldehyde; 0.045 – 2.84 mg m^{-3}) (Khamgaonkar and Fulare, cited in ATSDR, 1999).

Finally, ATSDR (1999) described one study suggesting changes in pulmonary functions in children exposed in the home (estimated 0.06 ppm formaldehyde; 0.075 mg m^{-3}). This study reported increased rates in bronchitis or asthma and decreased PEFR (Krzyzanowski *et al.*, cited in ATSDR, 1999). These changes were not observed in the adults. A second study of children schooled in particleboard schoolroom reported increased incidence of adverse effects associated with formaldehyde exposures in adult studies (rhinitis, cough, nose bleed, headaches) but at much lower exposure concentrations (0.075, 0.069, 0.043 ppm; 0.094, 0.086, 0.054 mg m^{-3}) (Wantke *et al.*, cited in ATSDR, 1999). These results may indicate that children are more sensitive to formaldehyde; however, the clinical significance of these results was "uncertain". The effects diminished when the children were removed (ATSDR, 1999).

4.4.1.2 Carcinogenic Effects

Over 30 cohort and case-control studies of human occupational exposures have been published as well as additional meta-analyses of reported data. The majority of these studies examine the potential impact on the respiratory tract. A few examined other non-respiratory carcinogenic outcomes; however, the results are not definitive and are not supported by formaldehyde's toxicokinetic and metabolic studies (*i.e.*, primarily deposited in the upper respiratory tract and rapidly metabolized) (EC, 2001, WHO, 2002). Details of a selected number of these reports are listed in Tables 10 and 11.

Although they had limited associations, a significant increase in nasopharyngeal cancer was observed in some occupational case-control studies (Vaughan *et al.*; Roush *et al.*, West *et al.*, cited in EC, 2001 and WHO, 2002); overall no increase was identified (Vaughan *et al.*, cited in EC, 2001 and WHO, 2002). Other studies reported no significant increases in nasal squamous cell carcinoma (Olsen and Anaes; Hayes *et al.*, Luc *et al.*, cited in EC, 2001 and WHO, 2002). A single study examining the association between adenocarcinoma in the nasal cavity reported a non-significant increase exacerbated (and possibly confounded) by concurrent exposure to wood dust (Luce *et al.*, cited in EC, 2001 and WHO, 2002). Examination of the available cohort studies also provided little evidence of nasopharyngeal cancers; problems such as the rarity of this cancer, the low numbers reported, and exposure to other chemicals/particulates were observed (EC, 2001 and WHO, 2002).

The majority of case-controlled studies did not report an association between exposure and lung cancer (EC, 2001; WHO, 2002). A cohort study observed a small, but significant increase in lung cancer (Blair *et al.*, cited in EC, 2001 and WHO, 2002); however, these results were not observed in the other cohort studies (Blair *et al.*; Blair *et al.*; Marsh *et al.*; Marsh *et al.*; Blair and Stewart; Callas *et al.*, EC, 2001 and CIADS, 2002).

Blair *et al.* and Partanen *et al.*, (cited in EC, 2001 and WHO, 2002) conducted a meta-analysis of epidemiological data from 1975-1991. No significant increase in risk of nasal cancer was reported by Blair *et al.*, whereas, Partanen *et al.*, reported an association for sinonasal cancer with "substantial" formaldehyde exposure (EC, 2001; CIADS, 2002). An increased risk of nasopharyngeal cancer was reported in high exposure groups (Blair *et al.*; Partanen *et al.*, cited in EC, 2001 and WHO, 2002). A small but significant increased risk of lung cancer was reported among exposed industrial workers, but not among the professional group (Blair *et al.*; Partanen *et al.*, cited in EC, 2001 and WHO, 2002). A more recent meta-analysis of available epidemiological data published between 1975-1995 found no increased risk with exposure and no risk of death due to nasopharyngeal cancer (possibly due to newer data not included in the earlier analyses and different interpretation of the older studies) (Collins *et al.*, cited in EC, 2001 and WHO, 2002).

In a 1995 assessment IARC classified formaldehyde as *Group 2A (probably carcinogenic to humans)*; IARC (2004) re-classified formaldehyde as *Group 1 (carcinogenic to humans)*. The current higher classification was based on information published since 1995 which reported a significant increase in death due to nasopharyngeal deaths in exposed workers; a strong association between exposure and risk of leukemia (but "not sufficient evidence for causal association"); and, limited evidence of an increase risk of sinonasal cancer (IARC, 2004). The U.S. EPA classifies formaldehyde as a *B1 - probable human carcinogen* (1991).

4.4.1.3 Other Effects

No reports of death associated with formaldehyde inhalation were identified (ATSDR, 1999; EC, 2001; CICAD, 2002).

As discussed in the Acute Sections, formaldehyde is an eye irritant; no other ocular effects were reported after chronic inhalation.

Some reports of skin irritation were made after occupational exposures; however, in most cases there was also direct dermal contact (Meding and Swanbeck; Menné *et al.*; Kiec-Swierczynska; Eberlein-Konig *et al.*, cited in ATSDR, 1999). Controlled inhalation studies found no dermal effects with exposure up to 3 ppm (3.75 mg m^{-3}) (Andersen and Molhave; Bender *et al.*, Day *et al.*, Gorski *et al.*, Krakowiak *et al.*; Kulle *et al.*; Pazdrak *et al.*, Weber-Tschopp *et al.*, cited in ATSDR, 1999).

There was limited evidence of adverse immunological effects with formaldehyde inhalation (ATSDR, 1999).

No significant effects on other systems (cardiovascular, gastrointestinal, haematological, musculoskeletal, hepatic, renal, endocrine, reproductive, developmental, body weight) were reported (ATSDR, 1999, EC, 2001).

4.4.2 Subchronic and Chronic Animal Effects

Tables 12 and 13 lists some examples of the lowest and highest NOAELs (No Observable Adverse Effect Level) and LOAELs (Lowest Observable Adverse Effect Level) reported in the literature.

4.4.2.1 Death

Long-term (sub-chronic and chronic) formaldehyde inhalation has been reported to significantly increase rate of mortality in rats and hamsters at exposures around 10-15 ppm (12.5-19 mg m^{-3})(Kamata *et al.*; Maronpot *et al.*; Albert *et al.*; Monticello *et al.*; Swenberg *et al.*, Kerns *et al.*; Dalbey; cited in ATSDR, 1999).

4.4.2.2 Respiratory Effects

As with the acute and sub-acute effects, chronic formaldehyde inhalation appears to affect most significantly the upper respiratory tissues with which there is direct contact; primarily the nasal epithelium.

Over 12 long-term (sub-chronic and chronic) laboratory animal (rats, mice, monkeys) studies were described in EC (2001) and CICAD (2001) which reported significant histological changes in the nasal epithelium after sub-chronic and chronic inhalation. ATSDR (1999) described over 23 long-term inhalation studies which reported histological changes in the nasal epithelium in rats, mice, and hamsters and changes in the nasal epithelium and in epithelium lower down the respiratory tract in monkeys. The majority of the animal studies documented were conducted on rats (ATSDR, 1999; EC, 2001; CICAD, 2002).

Table 8 NOAELs and LOAELs for Subchronic Formaldehyde Inhalation (Experimental Animals)

Effects Reported	Exposure Period	Air Concentration [b] ppm (mg m^{-3})	Species	Reference
Death:				
80% mortality. Serious LOAEL.	6 hr/d, 5 d/wk, 13 wk	40 (50)	Mice	Maronpot *et al.*, cited in ATSDR, 1999.
Systemic Effects: Respiratory Tract				
Histopathological effects in the nasal cavity. NOAEL.	6 hr/d, 5 d/wk, 13 wks	1 (1.2)	Rats	Woutersen *et al.*, cited in ATSDR, 1999 and EC, 2001).
Histopathological effects and cell proliferation in the nasal cavity. NOAEL.	6 hr/d, 5 d/wk, 11 wk + 4d	2.0 (2.4)	Male rats.	Casanova *et al.*, cited in ATSDR, 1999 and EC, 2001.
Increased DNA-protein crosslinkage in lateral, medial & posterior meatuses of the nose. NOAEL.	6 hr/d, 5 d/wk, 81 d	2.0 (2.4)	Male rats.	Casanova *et al.*, cited in ATSDR, 1999.
Histopathological effects (metaplasia, with keratinisation of the epithelial lining the larynx; cell turnover, squamous metaplasia & hyperplasia in the nasal turbinates). Serious LOAEL.	6 hr/d, 5 d/wk, 13 wks	9.7 (11.6)	Rat	Woutersen *et al.*, cited in ATSDR, 1999 and EC, 2001).
Histopathological effects and cell proliferation in the nasal cavity. Less Serious LOAEL.	6 hr/d, 5 d/wk, 11 wk + 4d	5.9 (7.1)	Male rats.	Casanova *et al.*, cited in ATSDR, 1999 and EC, 2001.
Increased DNA-protein crosslinkage in lateral, medial & posterior meatuses of the nose. Less Serious LOAEL.	6 hr/d, 5 d/wk, 81 d	6 (7.5)	Male rats.	Casanova *et al.*, cited in ATSDR, 1999.
Histopathological effects in the nasal epithelium and to a lesser extent in the larynx/trachea/carina epithelium. Serious LOAEL.	6 hr/d, 5 d/wk, 6 wk	6 (7.5)	Monkey	Monticello *et al.*, cited in ATSDR, 1999.
Mild ocular effects. Less serious LOAEL.		6 (7.5)	Monkey	Monticello *et al.*, cited in ATSDR, 1999.
Systemic Effects: Other				
Cardiovascular, gastrointestinal; haematological; hepatic, endocrine, ocular. NOAEL.	6 hr/d, 5 d/wk, 13 or 52 wk	10 (12.5)	Male rat	Appelman *et al.*, cited in ATSDR 1999.
Renal; body weight. NOAEL.	6 hr/d, 5 d/wk, 13 or 52 wk	1 (1.2)	Male rat	Appelman *et al.*, cited in ATSDR 1999.
Renal (increased incidence of oliguria); body weight (10% decrease). Less serious LOAEL.	6 hr/d, 5 d/wk, 13 or 52 wk	10 (12.5)	Male rat	Appelman *et al.*, cited in ATSDR 1999.

Table 12 NOAELs and LOAELs for Subchronic Formaldehyde Inhalation (Experimental Animals) (continued)

Effects Reported	Exposure Period	Air Concentration [b] ppm (mg m^{-3})	Species	Reference
Immunological/Lymphoreticular:				
Cell or humoral-mediated response. NOAEL.	6 hr/d, 5 d/wk, 3 wk	1 - 15 (1.2 - 18)	Rats and Mice	Dean *et al.*, Adams *et al.*, Holmstrom *et al.*, cited in EC, 2001.
Increased bacterial pulmonary survival. LOAEL.	Not defined	15 (18)	Mice	Jakab, cited in EC, 2001.
Increased ability of macrophages to release reactive oxygen intermediates. Less Serious LOAEL.	6 hr/d, 5 d/wk, 3 wk	15 (18)	Female mice	Adams *et al.*, cited in ATSDR, 1999.
NOAEL.	6 hr/d, 5 d/wk, 13 or 52 wk	10 (12.5)	Rats	Appelman *et al.*, cited in ATSDR, 1999.
NOAEL.	6 hr/d, 5 d/wk, 13 wk	20 (25)	Rats	Wouterson *et al.*, cited in ATSDR, 1999.
NOAEL	6 hr/d, 5 d/wk, 13 wk	40 (50)	Mice	Maronpot *et al.*, cited in ATSDR, 1999.
NOAEL	6 hr/d, 5 d/wk, 6 wk	6 (7.5)	Monkeys	Monticello *et al.*, cited in ATSDR, 1999.
Neurological:				
NOAEL.	6 hr, 5 d/w 13 or 52 wk.	10 (12.5)	Male rats	Appelman *et al.*, cited in ATSDR, 1999.
NOAEL	6 hr/d, 5 d/wk, 13 wk	10 (12.5)	Rats	Woutersen *et al.*, cited in ATSDR, 1999.
NOAEL	6 hr/d, 5 d/wk, 13 wk	10 (12.5)	Mice	Maronpot *et al.*, cited in ATSDR, 1999.
Temporary uncoordinated movement & wall climbing. Less Serious LOAEL.	6 hr/d, 5 d/wk, 13 wk	20 (25)	Rats	Woutersen *et al.*, cited in ATSDR, 1999.
Listless, hunched appearance. Less Serious LOAEL.	6 hr/d, 5 d/wk, 13 wk	20 (25)	Mice	Maronpot *et al.*, cited in ATSDR, 1999.
Ataxia. Serious LOAEL.	6 hr/d, 5 d/wk, 13 wk	40 (50)	Mice	Maronpot *et al.*, cited in ATSDR, 1999.

Table 12 NOAELs and LOAELs for Subchronic Formaldehyde Inhalation (Experimental Animals) (continued)

Effects Reported	Exposure Period	Air Concentration [b] ppm (mg m^{-3})	Species	Reference
Reproductive:				
NOAEL	6 hr/d, 5 d/wk, 13 wk	20 (25)	Female mice	Maronpot *et al.*, cited in ATSDR, 1999.
NOAEL	6 hr/d, 5 d/wk, 13 wk	40 (50)	Male mice	Maronpot *et al.*, cited in ATSDR, 1999.
Decreased prominence of endometrial glands & stroma: decrease in ovarian luteal tissue. Less Serious LOAEL.	6 hr/d, 5 d/wk, 13 wk	40 (50)	Female mice	Maronpot *et al.*, cited in ATSDR, 1999.
Cancer:				
Nasal tumours; squamous cell carcinoma & polypoid adenoma. Serious LOAEL.	6 hr/d, 5 d/wk, 4 wk	20 (25)	Male rats	Feron *et al.*, cited in ATSDR, 1999.
Nasal tumours; squamous cell carcinoma, cystic squamous cell carcinoma, carcinoma in situ and meloblastoma. Serious LOAEL.	6 hr/d, 5 d/wk, 13 wk	10 (12.5)	Male rats	Feron *et al.*, cited in ATSDR, 1999.

[a] NOAEL, Less serious LOAEL, and Serious LOAEL as identified by ATSDR (1999).

[b] When both units of concentration were not provided in the literature, the following conversion factor and assumptions were used: mg m^{-3} x 24.45/MW = ppm; MW = 30.03; air at 25°C and 101.3 kPa (760mmHg) (Plog *et al.*, 1996).

[c] gd = gestational days.

Table 9 NOAELs and LOAELs for Chronic Formaldehyde Inhalation (Experimental Animals)

Effects Reported	Exposure Period	Air Concentration [b] ppm (mg m^{-3})	Species	Reference
Death:				
Significantly reduced survival after 9 months. Serious LOAEL.	6 hr/d, 5 d/wk, 9 mo	15 (19)	Male rat	Kamata *et al.*, cited in ATSDR, 1999.
38% mortality. Serious LOAEL.	6 hr/d, 5 d/wk, 588 d (≈19 mo.)	14.2 (17.7)	Male rat	Albert *et al.*, cited in ATSDR, 1999.
Decreased survival rate. Serious LOAEL.	6 hr/d, 5 d/wk, 24 mo	15 (19)	Male rat	Monticello *et al.*, cited in ATSDR, 1999.
Significantly reduced survival after 17 months. Serious LOAEL.	6 hr/d, 5 d/wk, 24 mo	5.6 (7.0)	Male rat	Swenberg *et al.*; Kerns *et al.*, cited in ATSDR, 1999.
Significantly reduced survival after 12 months. Serious LOAEL.	6 hr/d, 5 d/wk, 24 mo	14.3 (17.9)	Male and female rats	Swenberg *et al.*; Kerns *et al.*, cited in ATSDR, 1999.
Significantly reduced survival times. Serious LOAEL.	5 hr/d, 5 d/wk, lifetime	10 (12.5)	Male hamster	Dalbey *et al.*, cited in ATSDR, 1999.
Systemic Effects: ***Respiratory Tract***				
Histopathological effects in the nasal cavity. NOAEL.	6 hr/d, 5 d/wk, 13 or 52 wk	1 (1.2)	Male rat	Appelman *et al.*, cited in ATSDR 1999 and EC, 2001.
Histopathological effects in the nasal cavity. NOAEL.	22 hr/d, 7 d/wk, 26 wk	0.98 (1.2)	Rats and monkeys	Rusch *et al.*, Cited in ATSDR, 1999.
Histopathological effects in the nasal cavity. NOAEL.	22 hr/d, 7 d/wk, 26 wk	1 (1.2)	Rats	Rusch *et al.*, Cited in EC,2001.
Histopathological effects and increased cell proliferation in the nasal cavity. NOAEL.	6 hr/d, 5 d/wk, up to 24 mo.	2 (2.4)	Male rats	Monticello *et al.*, cited in EC, 2001.
Histopathological effects in the nasal cavity. NOAEL.	6 hr/d, 5 d/wk, up to 28 mo.	0.3 (0.4)	Male rats	Kamata *et al.*, cited in EC, 2001.
Histopathological effects, in the nasal cavity. NOAEL.	6 hr/d, 5 d/wk, up to 24 mo.	2.0 (2.4)	Mice	Swenberg *et al*; Kerns *et al.*, cited in EC, 2001.
NOAEL.	22 hr/d, 7 d/wk, 26 wk	2.95 (3.6)	Hamster	Rusch *et al.*, Cited in ATSDR, 1999.

Table 13 NOAELs and LOAELs for Chronic Formaldehyde Inhalation (Experimental Animals) (continued)

Effects Reported	Exposure Period	Air Concentration [b] ppm (mg m^{-3})	Species	Reference
NOAEL.	6 hr/d, 5 d/wk, 13 wk	1 ppm (1.2)	Rats	Zwart *et al.*, cited in ATSDR, 1999 and EC 2001.
Histological changes to a small area in the nose. LOAEL.	6 hr/d, 5 d/wk, 13 wk	3 ppm (3.7)	Rats	Zwart *et al.*, cited in ATSDR, 1999 and EC 2001.
Histopathological effects in the nasal cavity (rhinitis; hyperplasia and metaplasia of nasal epithelium). Serious LOAEL.	6 hr/d, 5 d/wk, 13 or 52 wk	10 (12.5)	Male rat	Appelman *et al.*, cited in ATSDR 1999 and EC, 2001.
Histopathological effects in the nasal cavity (nasal squamous cell carcinoma & hyperplasia; basal cell hyperplasia; rhinitis). Serious LOAEL.	22 hr/d, 7 d/wk, 26 wk	2.95 (3.69)	Rats	Rusch *et al.*, Cited in ATSDR, 1999.
Histopathological effects in the nasal cavity. LOAEL.	22 hr/d, 7 d/wk, 26 wk	3 (3.6)	Rats and monkeys	Rusch *et al.*, Cited in EC,2001.
Histopathological effects, in the nasal cavity. LOAEL.	6 hr/d, 5 d/wk, up to 24 mo.	2.0 (2.4)	Rats	Swenberg *et al*; Kerns *et al.*, cited in EC, 2001.
Histopathological effects in the nasal cavity (hoarseness; nasal congestions and discharge; squamous metaplasia & hyperplasia in the nasoturbinates). Serious LOAEL.	22 hr/d, 7 d/wk, 26 wk	2.95 (3.69)	Male monkeys	Rusch *et al.*, Cited in ATSDR, 1999.
Histopathological effects and increased cell proliferation in the nasal cavity. NOAEL.	6 hr/d, 5 d/wk, up to 24 mo.	6 (7.2)	Male rats	Monticello *et al.*, cited in EC, 2001.
Histopathological effects in the nasal cavity. NOAEL.	6 hr/d, 5 d/wk, up to 28 mo.	2.17 (2.6)	Male rats	Kamata *et al.*, cited in EC, 2001.
Systemic Effects: Other				
Cardiovascular, gastrointestinal; haematological; hepatic, endocrine, ocular. NOAEL.	6 hr/d, 5 d/wk, 13 or 52 wk	10 (12.5)	Male rat	Appelman *et al.*, cited in ATSDR 1999.

Table 13 NOAELs and LOAELs for Chronic Formaldehyde Inhalation (Experimental Animals) (continued)

Effects Reported	Exposure Period	Air Concentration [b] ppm (mg m^{-3})	Species	Reference
Renal; body weight. NOAEL.	6 hr/d, 5 d/wk, 13 or 52 wk	1 (1.2)	Male rat	Appelman *et al.*, cited in ATSDR 1999.
Renal (increased incidence of oliguria); body weight (10% decrease). Less serious LOAEL.	6 hr/d, 5 d/wk, 13 or 52 wk	10 (12.5)	Male rat	Appelman *et al.*, cited in ATSDR 1999.
Body weight. NOAEL.	22 hr/d, 7 d/wk, 26 wk	2.95 (3.6)	Female rats	Rusch *et al.*, Cited in ATSDR, 1999.
Body weight. NOAEL.	22 hr/d, 7 d/wk, 26 wk	0.98 (1.22)	Male rats	Rusch *et al.*, Cited in ATSDR, 1999.
Body weight (average 13% decrease). Less Serious LOAEL.	22 hr/d, 7 d/wk, 26 wk	2.95 (3.6)	Male rats	Rusch *et al.*, Cited in ATSDR, 1999.
Body weight. NOAEL.	22 hr/d, 7 d/wk, 26 wk	2.95 (3.6)	Monkey	Rusch *et al.*, Cited in ATSDR, 1999.
Cancer:				
NOAEL.	6 hr/d, 5 d/wk, up to 24 mo.	5.6 (6.7)	Mice Hamsters	Kerns *et al.*, cited in EC, 2001.
NOAEL.	6 hr/d, 5 d/wk, up to 24 mo.	2 (2.4)	Male rats	Monticello *et al.*, cited in EC, 2001.
NOAEL	Life-time.	9.6 (12)	Hamsters	Dalbey, cited in EC, 2001.
Squamous cell carcinomas (10/100 rats). Serious LOAEL.	6 hr/d, 5 d/wk, 588 d (=19.5 mo)	14.2 (17.8)	Male rats	Albert *et al.*, cited in ATSDR, 1999.
Nasal squamous cell carcinomas (13/32 rats). Serious LOAEL.	6 hr/d, 5 d/wk, 28 mo.	15 (18.7)	Rats	Kamata *et al.*, cited in ATSDR, 1999 and EC, 2001.
Nasal tumours (20/90 rats). Serious LOAEL.	6 hr/d, 5 d/wk, 24 mo.	10 (12.5)	Male rats	Monticello *et al.*, cited in ATSDR, 1999.

Table 13 NOAELs and LOAELs for Chronic Formaldehyde Inhalation (Experimental Animals) (continued)

Effects Reported	Exposure Period	Air Concentration [b] ppm (mg m^{-3})	Species	Reference
Nasal tumours (20/90 rats). Serious LOAEL.	6 hr/d, 5 d/wk, 24 mo.	10 (12.5)	Male rats	Monticello *et al.*, cited in ATSDR, 1999.
Nasal cavity tumours: squamous cell carcinomas (38/100), fibrocarcinoma (1/100), mixed carcinoma (1/100). Serious LOAEL.	6 hr/d, 5 d/wk, lifetime	14.8 (17.8)	Male rats	Sellakumar *et al.*, cited in ATSDR, 1999 and EC, 2001.
Nasal squamous cell carcinoma (14/32 rats). LOAEL.	6 hr/d, 5 d/wk, 28 mo	14 (17)	Male rats	Tobe *et al.*, cited in EC, 2001.
Squamous cell carcinomas of the nasal cavity (106/235 rats). Serious LOAEL.	6 hr/d, 5 d/wk, 24 mo	14.3 (17.9)	Rats	Swenberg *et al.*, Kerns *et al.*, cited in ATSDR, 1999 and EC, 2001.

[a] NOAEL, Less serious LOAEL, and Serious LOAEL as identified by ATSDR (1999).

[b] When both units of concentration were not provided in the literature, the following conversion factor and assumptions were used: mg m^{-3} x 24.45/MW =ppm; MW=30.03, air at 25°C and 101.3 kPa (760mmHg) (Plog *et al.*, 1996).

The majority of the sub-chronic rat studies report formaldehyde induced histological effects and cell proliferation at concentrations greater than 3.7 mg m^{-3} (3 ppm); effects included hyperplasia, squamous cell metaplasia, inflammation, erosion, ulceration, and disarrangement of the nasal epithelium as well as cell proliferation in the nasal cavity (EC, 2001). With the exception of a few studies reporting some cell proliferation (Swenberg *et al.*; Zwart *et al.*, cited in HC/HC, 2001), no significant effects were observed at 1.2 or 2.4 mg m^{-3} (0.96 or 1.9 ppm) (EC, 2001). Review of sub-chronic animal exposures indicated that:

- rats and monkeys appear to be more sensitive to nasal damage than mice and hamsters (Maronpot *et al.*, cited in ATSDR, 1999);
- damage to the respiratory epithelium in monkeys is more widely distributed than in rats (Monticello *et al.*; Monticello *et al.*, cited in ATSDR, 1999) most likely due to differences in respiratory physiology; these changes occur in regions with high rates of cellular proliferation (Casanova *et al.*, cited in ATSDR, 1999);
- duration of exposure is less significant to respiratory tissue damage than exposure concentration (Wilmer *et al.*, cited in ATSDR, 1999. Swenberg *et al.*; Swenberg *et al.*; Wilmer *et al.*, Wilmer *et al.*, cited in EC, 2001); and,
- in rats nasal tissues, the number of formaldehyde-induced DNA-protein cross links occurs in the same regions damaged by formaldehyde (Cavanova *et al.*, cited in ATSDR, 1999).

The predominant non-carcinogenic effects reported in the chronic inhalation studies were histological changes to the nasal cavity and respiratory tract (primarily upper). In rats, the tissue most effected were the nasal epithelium in the anterior regions of the nasal cavity (Kamata *et al.*; Kerns *et al.*; Monticello *et al.*; Swenberg *et al.*; Woutersen *et al.*, cited in ATSDR, 1999). Rat

studies demonstrated non-neoplastic histological changes occurred at concentrations of 2.4 mg m^{-3} (1.9ppm) and neo-plastic lesions at 6 ppm (7.2 mg m^{-3}) and above (Swenberg *et al.*, Kerns *et al.*, Rusch *et al.*, Appelman, *et al.*, Woustersen *et al.*, Monticello *et al.*, cited in ATSDR, 1999 and EC, 2001).

In mice and hamsters, non-neoplastic damage to upper respiratory tract epithelium was observed after chronic exposures of 5.6 ppm (7.0 mg m^{-3}) and 10 ppm (12.5 mg m^{-3}) respectively (Kerns *et al.*; Dalbey, cited in ATSDR, 1999).

4.4.2.3 Immunological/Lymphoreticular

One inhalation study was identified (specific of exposures were not provided) which reported an immunological effect (Jakab, cited in EC, 2001); three studies identified no immunological effects (Dean *et al.*, Adams *et al.*, Holmstrom *et al.*, cited in EC, 2001).

ATSDR (1999) listed four long-term (sub-chronic and chronic) all of which did not identify an immunological response (Monticello *et al.*, Appelman *et al.*, Woutersen *et al.*, Maronpot *et al.*, cited in ATSDR, 1999). A single study in mice observed changes in macrophages after exposure to 15 ppm (19 mg m^{-3}) (Adams *et al.*, cited in ATSDR, 1999).

4.4.2.4 Cancer

A number of rat studies reporting carcinogenic effects in animals were identified (Albert *et al.*, Kamata *et al.*, Monticello *et al.*, Morgan *et al.*, Kerns *et al.*, Sellakumar *et al.*, Tobe *et al.*, cited in ATSDR, 1999 and EC, 2001). The exposure-response for all the studies were similar (highly non-linear); tumours (primarily nasal squamous cell carcinoma) were observed only at high exposures (6 ppm (7.2 mg m^{-3}) and above) (EC, 2001).

Nasal tumours were observed in mice exposed chronically to 14.3 ppm (17.9 mg m^{-3}) (Kerns *et al.*, cited in ATSDR, 1999). Hamsters exposed to 12 mg m^{-3} (9.6 ppm) did not have a carcinogenic effect (Dalbey, cited in EC, 2001).

Although formaldehyde is weakly genotoxic, can produce DNA-protein crosslinks in cell with direct contact (see Section 4.2), and causes tumours in rats, its carcinogenicity is more likely due to its cytotoxic properties (EC, 2001; OECD, 2002).

4.4.2.5 Other Effects

No effects were reported in the cardiovascular, gastrointestinal; haematological; hepatic, endocrine, or ocular systems in rats exposed sub-chronically to 10 ppm (12.5 mg m^{-3}); a slight change in body weight and the renal system was observed at this exposure, which was not observed at 1 ppm (1.2 mg m^{-3}) (Appelman *et al.*, cited in ATSDR, 1999). Some reduction in body weight was reported in male rats (effect not seen in females or monkeys) chronically exposed to 2.9 ppm (3.6 mg m^{-3}) (Rusch *et al.*, cited in ATSDR, 1999).

5.0 EFFECTS ON VEGETATION

The biological effects of formaldehyde on terrestrial vegetation were reviewed. A limited number of reports were identified following a search of the Web of Science database and were reviewed for the phytotoxicity of formaldehyde. Data from the Canadian Environmental Protection Act Priority List Substance Assessment Report for Formaldehyde (EC, 2001) were also reviewed and summarised below.

Formaldehyde is a toxic compound produced during plant one-carbon metabolism (Cossins, 1987). It is thought that formaldehyde in vivo is bound to endogenous nucleophiles, such as glutathione or tetrahydrofolate (Sardi and Tyihak, 1994; Chen *et al.*, 1997). There were limited reports on the phytotoxicity of formaldehyde (fumigation) on plants. The reports that were found are summarized below.

Mutters *et al.* (1993) exposed common Bean (*Phaseolus vulgaris*) cultivar UI-114 to formaldehyde at gaseous concentrations of 78, 128, 239, and 438 $\mu g\ m^{-3}$. Plants were exposure for 7 hours a day, 3 days a week, for a 4 week period. Treatment commenced on the emergence of floral bud. No adverse visible effects were reported (chlorosis or necrosis), the plants displayed increase leaf area and stem dry weight.

Muir and Shirazi (1997) evaluated the effect of simulated fogwater enriched with formaldehyde on Douglas fir (*Psuedotsuga menziiesii* (Mirbel) *franco*). The plants were treated with formaldehyde at concentrations of 3.6, 18 and 36 $\mu g\ m^{-3}$ for 6 hours/day over a 4 day period. The observed effects were delay in budbreak and slight decrease in height growth with no effect on root:shoot ratios. These effects were not statistically significant. In this study the authors also evaluated nitrogenase activity and growth diameter of the lichen, *Lobararia pulmonatia* L. Hoffm. Nitrogenase activity was slightly repressed (not significant) and growth rate was unaffected.

Masaru *et al.*, (1976) reported that exposure of formaldehyde in vivo reduces pollen tube length in lily (*Lilium longiflorum*). Formaldehyde levels used in this study were 440 and 1680 $\mu g\ m^{-3}$. Exposure time of 5 hours, at formaldehyde level of 1680 $\mu g\ m^{-3}$ resulted in loss of pollen tube elongation (loss of viability of pollen), whereas, exposure to 440 $\mu g\ m^{-3}$ for 1-2 hours resulted in a 50-60% reduction. These concentrations are dramatically higher than concentration found in polluted areas.

Shirazi and Muir (1998) reported that formaldehyde concentrations of, 300, 600, 900 and 1200 $mmol\ m^{-2}$, reduced germination of Douglas fir pollen. Pollen was treated with formaldehyde for 20 hours.

Haagen-Smit *et al.*, (1952) evaluated the effect of exposure to a formaldehyde concentration of 840 $\mu g\ m^{-3}$ for 5 hours on the plant species alfalfa (*Medico sativa*), spinach (*Spinacia oleracea*), beets (*Beta vulgaris*), or oats (*Avena sativa*). Alfalfa demonstrated mild atypical signs of injury at this formaldehyde concentration, whereas no injury was observed for spinach, beets and oats.

Giese *et al.*, (1994) evaluated the effect of gaseous formaldehyde on spider plant and two tobacco cultivars. The whole plant studies involved short term exposures (5 hours) to formaldehyde at 62, 420, or 4200 µg m^{-3} levels. Spider plants displayed no visible damage at the three concentration ranges, whereas, both tobacco cultivars when treated with 4200 µg m^{-3} were significantly damaged (>60% damaged leaf area). At the concentration of 420 µg m^{-3} one of the cultivars (Bel B) had small lesions that developed into necrotic spots (10% damaged leaf area). In addition, Giese *et al.*, (1994) evaluated the effect of formaldehyde on biomass of Soybean cell cultures. Soybean suspension cell culture was treated the with formaldehyde concentrations of 10^{-6}, 10^{-5}, 10^{-4}, 10^{-3} M for 5 days. The 10^{-3} M treatment resulted in a 48% decrease in cell dry weight.

The study by Barker and Shimabuku (cited in EC, 2001) evaluated the effect of formaldehyde (0, 18, and 54 µg m^{-3}) on aspen (*Populus tremuloides* Michx), winter wheat seedlings (*Triticum aestivum* L. Stephanes), rapeseed (*Brassica rapa* L. CrGc.1-8) and slash pine (*Pinus elliotti*). Plants were exposed for a period of 4.5 h per night, 3 times per week, for 40 days. Wheat seedlings and aspen were not affected by the formaldehyde exposure. A significant decrease was observed for leaf area, leaf dry weight, stem dry weight, flower number, and number of mature siliques (seed pods) when compared with control plants. An increase in needle and stem growth in slash pine was observed at these levels of formaldehyde.

6.0 EFFECT ON MATERIALS

The result of a literature review undertaken to identify, collect and compile the appropriate information for effects of ambient formaldehyde on materials indicates that no such published literature exists. This is because once released in air, formaldehyde is quickly photodegraded (i.e., 1.71 days) and is readily biodegradable. Formaldehyde, therefore, does not have a residence time long enough in ambient air to have any direct effects on materials.

7.0 AIR SAMPLING AND ANALYTICAL METHODS

7.1 Reference Methods

Air sampling and analytical methods for formaldehyde used in practice by established agencies are reported. In general, standard air monitoring methods for formaldehyde are based on liquid impinger, coated-solid cartridge, canister, spectrometric, sorbent tube, or passive sampling approaches. Widely employed and accepted reference air monitoring methods for formaldehyde have been developed, tested and reported by the United States Environmental Protection Agency (US EPA), NIOSH, and OSHA. Refer to Table 14 for a description of individual method advantages and disadvantages.

7.1.1 US EPA Compendium Method TO-5

US EPA has developed a number of methodologies suitable for sampling ambient air for trace-level concentrations of formaldehyde. US EPA Compendium Method TO-5 describes the determination of individual aldehydes and ketones (including formaldehyde) in ambient air using liquid impinger sampling followed by analysis with high performance liquid chromatography (HPLC) with an ultraviolet (UV) detector (US EPA, 1999). Advantages of this method include: specific for aldehydes and ketones, good stability for derivative compounds formed in the impingers and relatively low detection limits (i.e., 1-50 ppbv). Disadvantages of this method include: formaldehyde laboratory contamination is common, sensitivity limited by reagent purity, potential for evaporation of liquid over long term sampling, and isomeric aldehydes and ketones may be unresolved by the HPLC system.

In this method, a low volume pump is used to draw ambient air through a midget impinger containing a solution of dinitrophenyl hydrazine (DNPH). The low volume pump is operated for 12 hours at a rate of 100 mL/min to collect a total volume of 72 L. After sampling, the impinger solution is placed in a screw-capped vial and returned to the laboratory for analysis. The DNPH derivatives are recovered by removing the isooctane layer, extracting the aqueous layer with 10 mL of 70/30 hexane/methylene chloride, and combining the organic layers. The combined organic layers are evaporated to dryness under a steam of nitrogen and the residue dissolved in methanol. The DNPH derivatives are determined using reversed phase HPLC with an UV adsorption detector operated at 370 nm.

7.1.2 US EPA Compendium Method TO-11A

US EPA Compendium Method TO-11A describes an active sampling methodology for the determination of formaldehyde and other carbonyl compounds in ambient air utilizing a DNPH coated-solid adsorbent followed by analysis with HPLC/UV detection (US EPA, 1999). The procedure is similar to US EPA Compendium Method TO-5 except that a coated silica gel cartridge is used instead of a liquid impinger. The advantages of this method include: placement of sorbent as first element in the sampling train minimizes contamination, sampling system is

portable and lightweight, large database, and proven technology. Disadvantages of this method include: isometric aldehydes and ketones and other compounds with the same HPLC retention time may interfere, liquid water captured on the DNPH cartridge during sampling may interfere, carbonyls on the DNPH cartridge may degrade if an ozone denuder is not employed, and ozone and UV light deteriorates trapped carbonyls on cartridge.

In this method, a known volume of ambient air is drawn through a prepackaged cartridge coated with acidified DNPH at a sampling rate of 100 to 2000 mL/min for an appropriate period of time. Sampling rate and time are dependent upon carbonyl concentration in the test atmosphere. After sampling, the sample cartridges and field blanks are individually capped and placed in shipping tubes with polypropylene caps. The capped tubes are placed in a polypropylene shipping container cooled to subambient temperature and returned to the laboratory for analysis. The cartridges are then removed from the vials and washed with acetonitrile by gravity feed elution. The eluate is diluted volumetrically and an aliquot is removed for determination of the DNPH-formaldehyde derivative by isocratic reverse phase HPLC with UV detection at 360 nm. Formaldehyde and other carbonyl compounds in the sample are identified and quantified by comparison of their retention times and peak heights or peak areas with those of standard solutions. Typically, carbonyl compounds are measured effectively to less than 0.5 ppbv.

7.1.3 US EPA Compendium Method TO-15A

US EPA Compendium Method TO-15A describes the determination of VOCs (including formaldehyde) in air collected in specially prepared canisters and analyzed by gas chromatography/mass spectrometry (GC/MS) (US EPA, 1999). The advantages of this method include: incorporates a multisorbent/dry purge technique or equivalent for water management thereby addressing a more extensive set of compounds, establishes method performance criteria for acceptance of data, provides enhanced provisions for quality control, and unique water management approach allows analysis for polar VOCs. Disadvantages of this method are that it requires expensive analytical equipment and a high level of operator skill to perform.

In this method, the ambient atmosphere is sampled by introduction of 6 L of air into a specially prepared stainless steel canister (SUMMA or equivalent) over an appropriate time and rate. Both subatmospheric pressure and pressurized sampling modes make use of an initially evacuated canister. A pump ventilated sampling line is used during sample collection with most commercially available samplers. Pressurized sampling requires an additional pump to provide positive pressure to the sample canister. A sample of air is drawn through a sampling train comprised of components that regulate the rate and duration of sampling into the pre-evacuated and passivated canister. After the air is collected the canister valve is closed, an identification tag is attached to the canister, and the canister is transported to the laboratory for analysis. Upon receipt at the laboratory the canister tag data is recorded and the canister is stored until analysis.

To analyze the sample, a known volume of sample is directed from the canister through a solid multisorbent concentrator. A portion of the water vapour in the sample breaks through the concentrator during sampling to a degree depending on the multisorbent composition, duration of sampling, and other factors. Dry purging the concentrator with helium while retaining target

compounds can further reduce water content of the sample. After the concentration and drying steps are completed, the VOCs are thermally desorbed, entrained in a carrier gas stream, and then focused in a small volume by trapping on a reduced temperature trap or a small volume multisorbent trap. The sample is then released by thermal desorption and carried onto a gas chromatographic column for separation.

The analytical strategy for US EPA Compendium Method TO-15A involves using a high-resolution gas chromatograph (GC) coupled to a mass spectrometer (MS). If the MS is a linear quadrupole system, it is operated either by continuously scanning a wide range of mass to charge ratios (SCAN mode) or by monitoring select ion monitoring mode (SIM) of compounds on the target list. If the MS is based on a standard ion trap design, only a scanning mode is used. Mass spectra for individual peaks in the total ion chromatogram are examined with respect to fragmentation pattern of ions corresponding to various VOCs including the intensity of primary and secondary ions. The fragmentation pattern is compared with stored spectra taken under similar conditions, in order to identify the compound.

For any given compound, the intensity of the primary fragment is compared with the system response to the primary fragment for known amounts of the compound. This establishes the compound concentration that exists in the sample. This method applies to ambient concentrations of VOCs above 0.5 ppbv and typically requires VOC enrichment by concentrating up to 1 L of a sample volume.

7.1.4 NIOSH Method 2016

In addition to the air monitoring methods for formaldehyde developed by the US EPA, both the NIOSH and the OSHA have also developed methods for formaldehyde that are suitable for occupational, personal and area monitoring. The first methodology used by the NIOSH to determine formaldehyde in air (NIOSH Method 2016) consists of collecting formaldehyde on a cartridge containing silica gel coated with 2,4-dinitrophenylhydrazine (DNPH) (NIOSH, 2003). The sample is extracted with carbonyl-free acetonitrile, eluted through the cartridge, and analyzed by HPLC with UV detection. Sampling is conducted by drawing air through the cartridge using a personal sampling pump. The suggested flow rate is 0.03 to 1.5 L/min to obtain a volume between 1 and 15 L. The working range is 0.015 to 2.5 mg/m^3 for a 15 L air sample. This method can be used for the determination of formaldehyde for both STEL and time weighted average exposures. The analysis procedure in this method has also been used for detection of formaldehyde in automobile exhaust.

7.1.5 NIOSH Method 2541

A second methodology used by the NIOSH to determine formaldehyde in air (NIOSH Method 2541) consists of detecting concentrations of formaldehyde using gas chromatography (GC) (NIOSH, 1994a). Sampling is conducted by drawing air through a coated solid sorbent tube (XAD-2) using a sampling pump at a flow rate of 0.01 to 0.10 L/min to collect a volume between 1 and 36 L. The sample is subsequently desorbed with toluene, and analyzed using GC/FID

(flame ionization detection) or GC/NPD (nitrogen specific detection). The working range is 0.24 to 16 ppm (0.3 to 20 mg/m^3) for a 10 L air sample.

7.1.6 NIOSH Method 3500

A final methodology used by the NIOSH to determine formaldehyde in air (NIOSH Method 3500) is also the most sensitive of all the NIOSH methods. Sampling consists of collecting formaldehyde on a filter and two impingers containing a sodium bisulphate solution and analyzing with visible absorption spectrometry (VIS) (NIOSH, 1994b). Sampling is conducted by drawing air through the instrument at a flow rate of is 0.2 to 1 L/min. The air is subsequently analyzed via VIS and the results displayed. This method is able to measure ceiling levels as low as 0.1 ppm (15 L sample). It is best suited for the determination of formaldehyde in area samples. The working range is 0.02 to 4 ppm (0.025 to 4.6 mg/m^3) for an 80 L air sample.

7.1.7 OSHA Method 52

Occupational Safety and Health Administration has developed fully validated methods for the determination of formaldehyde that is suitable for occupational, personal and area monitoring. The first methodology used by the OSHA to determine formaldehyde in air (OSHA Method 52) (OSHA, 1989). This method consists of collecting formaldehyde by drawing a known volume of air through standard sized sampling tubes (containing XAD-2 adsorbent which has been coated with 2-(hydroxymethyl) piperidine) using a personal sampling pump. The suggested flow rate is 0.1 L/min and the recommended volume collected is 24 L. The samples are desorbed with toluene and then analyzed by gas chromatography using a nitrogen selective detector (GC/NPD). The target concentration for this method is 3 ppmv or 3.7 mg m^{-3}.

7.1.8 OSHA Method ID-205

The second methodology used by the OSHA to determine formaldehyde in air (OSHA Method ID-205) describes the collection of airborne formaldehyde in the breathing zone of workplace personnel and the subsequent analysis of those samples using a colorimetric technique (OSHA, 1990). Samples are collected on a passive badge monitor containing bisulfite-impregnated paper. The monitors are exposed to the atmosphere for 4 to 16 hours and then analyzed using a modified chromotropic acid procedure. Sample filters are desorbed using deionized water, acidified and chromotropic acid is added. The color complex formed is analyzed using a UV spectrophotometer at 580 nm. The qualitative detection limit of the overall procedure is 0.039 ppm at a 4 hour sampling duration. The quantitative detection limit of the overall procedure is 0.11 ppm at a 4 hour sampling duration.

7.2 Alternative, Emerging Technologies

Reports, journal articles, conference proceedings and other sources known to contain information on ambient measurement methods for chemicals such as formaldehyde were reviewed to determine the current status of alternative and emerging technologies. The result of the review indicates that much effort has been taken over the last few years to develop new technologies because of the central role formaldehyde plays in atmospheric chemistry. Several examples of alternative and emerging technologies have been developed and reported and many reviews been complied (Hak *et al.*, 2005; Aragon *et al.*, 2000; Cardenas *et al.*, 2000; Vairavamurthy *et al.*, 1992; Otson and Fellin, 1988). Based on these reviews, methods for the detection of formaldehyde in air can be put into general categories: chromatographic, spectroscopic, colorimetric, fluorimetric, chemiluminescent, and passive techniques. Refer to Table 14 for a description of individual category advantages and disadvantages.

7.2.1 Chromatographic Techniques

The most popular method for the determination of formaldehyde levels in air is based on the conversion of carbonyls to stable hydrazones when trapped in dinitophenyl hydrazine (DNPH) coated substrates. The hydrazones can then be separated by high performance liquid chromatography (HPLC) or gas chromatography (GC) and detected through various analytical techniques such as UV detection. These systems are highly suited for formaldehyde detection as they are selective and sensitive. They are also easy to set up in the field and are inexpensive. However, analytical detection is labour intensive and requires the use of wet chemical methods. Different versions of this technique involve unique reagents, alternate analytical techniques or different sampler designs.

Although the most widely used reagent for derivatization continues to be DNPH, others, such as sodium bisulphate, Girard's T reagent and various hydrazines and hydroxylamines, have been proposed (Vairavamurthy *et al.*, 1992). In addition, although HPLC remains the overwhelming choice for analysis, others have been used. For instance, GC can be used using several types of detectors. These measurements can be done directly or after derivatization with DNPH as with HPLC. The types of detectors used include: flame ionization detection (FID), thermal conductivity detection (TCD), electron capture detection (ECD) and mass spectrometry (MS) (Aragon *et al.*, 2000; Vairavamurthy *et al.*, 1992). Other examples of alternative analytical techniques that have shown some promise in the detection of formaldehyde include ultraviolet, 2-dimensional gas chromatographic, nebulization/reflux concentration, diode array and mass spectrometric detectors (Achatz *et al.*, 1999; Grosjean *et al.*, 1999; Sakuragawa *et al.*, 1999; Vairavamurthy *et al.*, 1992).

A new electrochromatography analytical method called capillary electrophoresis (CEC) was developed for the separation and quantitation of aldehydes and ketones (including formaldehyde) in ambient air (Fung and Long, 2001). In this method, air is sampled through a DNPH coated-solid cartridge in the field by a small air pump. The sample is subsequently desorbed and individual aldehydes and ketones are separated by electrochromatography and analyzed by mass

spectrometry. This method can detect formaldehyde in ambient air in concentrations as low as 0.041 $\mu g/m^3$.

Chromatographic techniques can employ two different sampling approaches: integrated or continuous. The majority of integrated sampling studies make use of liquid impingers which physically bubble the air sample through liquid to promote dissolution of the formaldehyde and the formation of less volatile hydrazones or other derivatives (Vairavamurthy *et al.*, 1992). Although impinger techniques have been used, they are cumbersome and are not well suited to large field studies at remote locations.

The use of solid sorbents techniques result in much higher sensitivity than the impinger method and are much more convenient to employ in the field as they are pre-concentrated. A number of solid sorbents have been made for this purpose: glass beads, glass fiber filters, silica gel, Chromosorb P, porous polymers, XAD-2, Florisil, and carbon (C_{18}) are the most common (Sandner *et al.*, 2001; Vairavamurthy *et al.*, 1992; Zhou and Mopper, 1990).

An emerging integrated method known as cryotrapping has also been developed which uses glass traps cooled in liquid nitrogen for monitoring carbonyl compounds in air (Levart and Verber, 2001). Sampling is followed by derivatization by DNPH with analysis by HPLC and detection by a diode array detector. Finally, a denuder tube coated with 2-hyroxymethylpiperidine (2-HMP) connected to a Tenax TA absorbent trap has been used as an integrated method (Thomas *et al.*, 1997). Formaldehyde is diffused to the walls of the denuder and reacted yielding a derivative which is trapped. The trap is thermally desorbed and then analyzed by GC-MS.

In order to allow for continuous, unattended measurement, new automatic instruments for the detection of formaldehyde have been developed. Automated continuously recording instruments with fast response times offer the best solution for monitoring average concentrations accurately, but most are bulky and expensive. In addition, many instruments are either insufficiently sensitive or selective in their measurements. An automated measurement system for aldehydes and ketones (including formaldehyde) using a diffusion scrubber in combination with HPLC was developed (Komazaki *et al.*, 1998; Possanzini *et al.*, 1996). Formaldehyde is effectively collected by the diffusion scrubber, which consists of a hydrophobic porous tube disposed concentrically within a Pyrex-glass tube and a DNPH scrubbing solution. After the collection of the gas sample, the sample solution in the diffusion scrubber is injected into the HPLC system and individual species are separated and determined. All measurement operations are sequenced by a programmable controller which allows automated continuous measurement to be performed at 10-minute intervals.

7.2.2 Spectroscopic Techniques

The previously described method uses indirect techniques to measure atmospheric formaldehyde concentrations through the two-stage process of sample collection and analysis. Direct formaldehyde measurements by optical techniques based on the spectroscopic properties of the formaldehyde molecule do not require this two-stage process. Advanced optical methods like

differential optical absorption spectroscopy (DOAS), Fourier Transform Infrared (FTIR) spectroscopy, laser-induced fluorescence (LIFS) and tunable diode laser absorption system (TDLAS) have recently been used to directly measure atmospheric concentrations of formaldehyde. The FTIR, LIFS and TDLAS techniques are based on the measurement of the absorbance due to absorption of infrared radiation by formaldehyde. The DOAS technique uses the same type of measurement but at the UV/VIS regions of the spectrum.

Operationally, these optical methods are capable of accurately following changes in formaldehyde levels over short time intervals with a very low detection limits and are non-destructive. With respect to the deployment and operation of these instruments there are some disadvantages. While the systems are automated, they cannot run unattended for long periods of time because the lasers need constant attention; they are very expensive; they are technically complex and use corrosive chemicals; they are fixed monitoring methods; and they require large amounts of electrical power. Most of these instruments, therefore, cannot at present be regarded as being suitable methods for routine ambient measurements in the field.

The use of optical methods to determine ambient formaldehyde concentrations is demonstrated by the differential optical absorption spectroscopy (DOAS) technique (Vairavamurthy *et al.*, 1992). The DOAS system is comprised of a light source, a receiver, a detection system and a computer. The light source is a high pressure Xenon lamp generating light in the wavelength region of 200 – 1000 nanometers (nm) which is placed 5 to 10 km form the detection device. The received light is transmitted by a fiber optic cable to the detection system, consisting of a spectrometer with a rapid scanning device and a computer. Averaged spectra are analyzed by a computer program based on known reference spectra of components absorbing in the measured spectral region. Formaldehyde concentrations are calculated from the measured and reference spectra.

Measurement of ambient formaldehyde can be carried out using an open multiple reflection optical system coupled to a Fourier Transform Infrared (FTIR) spectrometer (Li *et al.*, 2002; Tuazon *et al.*, 1978). A FTIR system is capable of operating at pathlengths up to 2 kilometers with the use of an eight-mirror multiple reflective cell within the 25 meter base-path. The formaldehyde concentrations are measured in the same fashion as the other spectroscopic techniques but this method has added high spectral resolution and good time resolution to the strengths of the other methods. The expense and bulk of this instrument; however, currently makes it an unrealistic choice for the measurement of formaldehyde concentrations in ambient air.

Laser-induced fluorescence (LIFS) has been used to detect formaldehyde (Vairavamurthy *et al.*, 1992). In this technique, a frequency doubled tunable dye laser or a pulsed laser can be used in a multipass cell for excitation of the formaldehyde. This method exhibits very poor detection limits (i.e., greater than 10 ppb) and is not very useful for ambient measurements.

Another direct spectroscopic technique is the tunable diode laser absorption system (TDLAS) which appears to be the best suited of the spectroscopic techniques for ambient measurements because advantages in sensitivity, selectivity and time resolution (Friedfeld *et al.*, 2000; Vairavamurthy *et al.*, 1992). The TDLAS employs a tunable diode laser to scan over a narrow

wavelength region for the particular absorption line of formaldehyde. Formaldehyde concentrations are determined by monitoring the extent of infrared absorption by the formaldehyde. The TDLAS system is currently able to detect concentrations as low as 0.25 ppb with a 3 minute time resolution. These systems have been used successfully in many ambient measurement programs (Li *et al.*, 2004; Sauer *et al.*, 2003; Fried *et al.* 2002).

7.2.3 Colorimetric Techniques

Several techniques have been used to determine formaldehyde colorimetrically including the chromotropic acid and modified pararosaniline method (Otson and Fellin, 1988). Some of these techniques can be automated (Vairavamurthy *et al.*, 1992) and can have adequate sensitivity and low blanks, but they are slow in developing color, require long sampling times and have some inferences.

In the chromotropic acid method, air is passed through a trap or impinger containing water, sodium bisulphate or alumina. The contents are mixed with sulphuric acid and chromotropic acid. Formaldehyde in the sample reacts to form a violet-coloured derivative which is determined spectrophotometrically. For sampling, an air sample is scrubbed into an aqueous solution by way of charged liquid impingers. This method is specific for formaldehyde but not very sensitive. The modified pararosaniline method has much improved sensitivity over the previous method but is prone to interferences from other carbonyl compounds. In this method, a solution of acidified pararosaniline reagent and mercury sulphite is added to the formaldehyde solution from the impinger. This leads to the formation of a purple color which can be determined spectrophotometrically.

Pretto *et al.* (2000) developed an alternative colorimetric technique to those above which allows for sensitive in situ measurements of formaldehyde in air. The formaldehyde is collected in a hanging drop of chromotropic acid and the resulting violet-pink dye is measured by an optical fiber/LED sensor. The device is inexpensive, simple and requires little operator care which makes this device a good choice for ambient measurements.

7.2.4 Fluorimetric Techniques

Determination of formaldehyde by fluorescence requires three steps: stripping of formaldehyde from the gas phase to the liquid phase, reaction into an easily measurable form, and then detection. The stripping of formaldehyde has been performed in different ways with fluorescent techniques. Automated diffusion scrubbers have been widely used for this purpose (Vairavamurthy *et al.*, 1992). These systems have many disadvantages; however, because they are likely to become clogged by particles in the air and growth of biota on the surface of the membrane. They also require continuous cleaning and are not easy to construct. Glass coils have also been used to strip formaldehyde from the gas phase to the liquid phase (Vairavamurthy *et al.*, 1992). In these devices, a stripping solution is pumped into the coil and forced into contact with the air sample.

Once in the liquid phase there are generally two fluorimetric techniques to detect formaldehyde. The Enzymatic technique consists of the oxidation of formaldehyde by nicotinamide adenine dinucleotide (NAD) and the reduction of NAD to its fluorescent form (Cardenas *et al.*, 2000). The reaction is catalyzed by an enzyme. This technique is free of interferes, automated, sensitive and fast. However, large amounts of expensive enzyme are required. Another technique is known as the Hantzsch reagent method (Cardenas *et al.*, 2000). This method is based on the observation that when formaldehyde is added to solutions containing acetylacetone and ammonium ions a yellow colour slowly develops. This colour is due to the formation of diacetlydihydrolutidine (DDL) which can be detected with fluorescence. This technique is very selective for formaldehyde.

7.2.5 Chemiluminescent Techniques

An additional method for the determination of ambient formaldehyde concentrations is based on the reaction of formaldehyde with gallic acid and hydrogen peroxide in a strongly alkaline solution to produce chemiluminescence (Vairavamurthy *et al.*, 1992). Samples are generally collected in a liquid impinger and then reacted later. The emissions produced by the chemiluminescence reaction are proportional to the formaldehyde concentration. This technique has recently been automated by the development of a continuous flow device (Maeda *et al.*, 1994). A negative effect caused by an unknown interferent is a major shortcoming of this method which makes this method unsuitable for field studies.

7.2.6 Passive Techniques

Techniques suitable for active sampling can normally be applied to passive sampling as well. In fact, for formaldehyde, considerable effort has been expended in the development of passive approaches. The advantages of these samplers are that there are cheap, simple to use, have no moving parts to break down, regular flow calibration is unnecessary, able to provide high spatial resolution, do not require power supplies, can be used to assess long-term concentrations and are well-suited for remote area measurements. However, only when low-level contaminants are present can reasonable time resolution be achieved. The sampler is exposed to ambient conditions for a set period of time (usually a much longer period than for active pump sampling) and then analyzed by an appropriate analytical method (Brown and Wright, 1994; Levin and Lindahl, 1994).

Uchiyama and Hasegawa (1999) have developed a diffusive sampling device for organic carbonyl compounds (including formaldehyde) which is suitable for the collection and analysis of low ppb concentration levels. This sampling device is composed of three parts, an exposure part made of a porous polytetrafluoroethylene (PPTFE) tube, an analysis part made of polypropylene tubing and an absorbent part made of DNPH coated silica gel. Formaldehyde is absorbed by the DNPH and subsequently detected by HPLC/UV.

Another example of a tube-type passive sampler has been reported which employs a DNSH-coated solid sorbent for the collection of formaldehyde and a sensitive HPLC-fluorescence

technique for its detection (Zhang *et al.*, 2000). This device can detect levels of aldehydes and ketones in the range from 0.4 to 1.6 ppb.

Muntuta-Kinyanta and Hardy (1991) developed a passive method with membrane permeation sampling for the determination of time weighted averages of formaldehyde in air. The sampling device is comprised of an unbacked dimethyl silicone membrane attached to the base of a glass tube. Formaldehyde permeates the membrane and reacts with a coated solid sorbent (XAD-2). Sampling times from 15 minutes to 8 hours have been reported (Muntuta-Kinyanta and Hardy, 1991). The derivative produced is thermally desorbed and determined by GC-FID. The detection limit for the method is 0.03 ppm for an 8 hours sampling period.

Table 10 Advantages and Disadvantages of Sampling and Analytical Methods

Method	Advantages	Disadvantages
US EPA TO-5	Specific for aldehydes and ketones Good stability for derivative compounds formed in the impingers Low detection limits (i.e., 1-50 ppbv)	Laboratory contamination is common Sensitivity limited by reagent purity Potential for evaporation of liquid over long term sampling Isomeric aldehydes and ketones may be unresolved by the HPLC system
US EPA TO-11A	Placement of sorbent as first element in the sampling train minimizes contamination Sampling system is portable and lightweight Large database Proven technology	Isometric aldehydes and ketones and other compounds with the same HPLC retention time may interfere Liquid water captured on the DNPH cartridge during sampling may interfere Carbonyls on the DNPH cartridge may degrade if an ozone denuder is not employed Ozone and UV light deteriorates trapped carbonyls on cartridge
U.S. EPA TO-15A	Addresses a large set of compounds Establishes method performance criteria for acceptance of data Provides quality control provisions Allows analysis for polar VOCs	Requires expensive analytical equipment Requires high level of operator skill
NIOSH Method 2016	NA	NA
NIOSH Method 2541	NA	NA
NIOSH Method 3500	NA	NA
OSHA Method 52	NA	NA
OSHA Method ID-205	NA	NA
Chromatographic Techniques	Most widely applied Simple to implement Good stability Specific and sensitive Inexpensive Can be automated	Subject to interferences Requires wet chemistry which is labour intensive
Spectroscopic Techniques	Directly allow for sensitive, rapid and quantitative formaldehyde detection Fully automated and well-suited for continuous measurements Lowest detection limits available Good time resolutions High spectral resolutions	Very expensive and bulky Require highly trained operators Use corrosive chemicals Fixed monitoring methods Require large amounts of power Cannot be run unattended for long periods
Colorimetric Techniques	Simple to implement Inexpensive Can be automated	Either too specific or not specific enough Insufficient sensitivity Subject to interferences
Fluorimetric Techniques	Can be automated Specific and sensitive	Subject to interferences System can become clogged Not easy to construct

Table 14 Advantages and Disadvantages of Sampling and Analytical Methods (continued)

Method	Advantages	Disadvantages
Chemiluminescent Techniques	Can be automated	Subject to interferences
Passive Techniques	Inexpensive and easy to use Provide high spatial resolution Do not require power supply Assess long-term concentrations Well-suited for remote monitoring No moving parts to break down Regular flow calibration unnecessary No bulky, expensive pumps required	Not often used for ambient monitoring Only reliable at higher ambient concentrations Long exposure times required

NA denotes not available.

8.0 AMBIENT OBJECTIVES IN OTHER JURISDICTIONS

Current and or recommended and proposed ambient guidelines of other jurisdictions in Canada, United States and elsewhere were reviewed for formaldehyde. Details about guidelines that exist for each jurisdiction reviewed are presented in tabular format in Appendix B. All jurisdictions have common uses for their guidelines. These uses may include:

- reviewing permit applications for sources that emit air pollutants to the atmosphere,
- investigating accidental releases or community complaints about adverse air quality for the purpose of determining follow-up or enforcement activity,
- determining whether to implement temporary emission control actions under persistent adverse air quality conditions of a short-term nature.

8.1 Formaldehyde Air Quality Guidelines and Objectives

Air quality guidelines for formaldehyde are summarized in Table 15. The principal approach by which guidelines are developed involves using carcinogenic risk assessment procedures. Pre-existing cancer risk assessments performed by others (e.g. US EPA Integrated Risk Information System summary data) are used to establish ambient air levels based on acceptable levels of lifetime cancer risk, such as one in 100,000 (10^{-5}) or one in 1,000,000 (10^{-6}). In only one case – Arizona Department of Environmental Quality (DEQ) (2005) – was the US OSHA 15-minute STEL of 2 ppm (2,456 µg m^{-3}) used to develop an ambient air guideline.

8.1.1 Canada

The derivation basis of air quality guidelines employed by Canadian agencies was, for the most part, unknown. Health Canada (2005) has developed residential indoor air quality guidelines of 123 µg m^{-3} (1-hour) and 50 µg m^{-3} (8-hour). Alberta Environment (2005) has a 1-hour Ambient Air Quality Objective of 65 µg m^{-3}, while the Ontario Ministry of the Environment (OME) uses the same value (65 µg m^{-3}) for ½-hour and 24-hour standards (OME, 2005).

British Columbia Ministry of Environment (2005) has 1-hour air Quality Objectives – 60 µg m^{-3} as an action level and 370 µg m^{-3} as an episode level. The action level is the target used when managing the level of formaldehyde in an airshed. The episode level corresponds to the concentration that starts to be of concern to public health (it is at this level that immediate steps be taken to reduce the release of formaldehyde into the atmosphere). Manitoba Conservation (2005) has a 1-hour criterion of 60 µg m^{-3}.

8.1.2 United States

The US Agency for Toxic Substances and Disease Registry (2005) adopted inhalation minimum risk levels of 49 µg m^{-3} (acute), 37 µg m^{-3} (intermediate), and 10 µg m^{-3} (chronic) based on

human exposure and animal study data. The US EPA (2005) uses an inhalation unit risk factor of 1.3E-05 per $\mu g\ m^{-3}$ based on observation of squamous cell carcinoma in a 2-year rat study and using the linearized multistage procedure. The inhalation unit risk factor is intended for use by US EPA staff in risk assessments, decision-making and regulatory activities.

Seven of the US agencies reviewed – Arizona, Massachusetts, Michigan, Minnesota, Rhode Island, Vermont, and Washington – have adopted or derived their 1-hour (in the case of Michigan) and annual average values from the US EPA inhalation unit risk factor. In only one case – Arizona DEQ (2005) – was and occupational exposure limit US OSHA 15-minute STEL of 2 ppm (2,456 $\mu g\ m^{-3}$)) used to develop an ambient air guideline.

Eighteen US states were reviewed for air quality guidelines:

- Four of these states – Indiana, New Jersey, Ohio, and Wisconsin – did not have guidelines.
- Seven states use a 1-hour guideline with values ranging from 15 to 150 $\mu g\ m^{-3}$
- Five states use a 24-hour guideline with values ranging from 0.33 to 40 $\mu g\ m^{-3}$
- Ten states use an annual guideline with values ranging from 0.08 to 7.69 $\mu g\ m^{-3}$

8.1.3 International Agencies

The World Health Organization WHO (2000) adopted a 30-minute reference exposure level of 100 $\mu g\ m^{-3}$ to prevent significant sensory irritation in the general population. The New Zealand Ministry of Environment and Ministry of Health (2002) adopted a health-based guideline value 100 $\mu g\ m^{-3}$ as a 30-minute average after WHO (2000). The Netherlands National Institute of Public Health (2001) does not have air quality criteria for formaldehyde.

8.2 Use of Occupational Limits for Ambient Air Quality Guidelines

Although widely practiced, there are several limitations in the direct and indirect application of occupational limits for ambient air quality guidelines:

- Occupational limits are based on the information gathered in workplace, through experience from medical research and practice, from experimental human and animal studies, and from a combination of these sources. Often they are based upon averaged tolerated doses from actual repeated industrial exposures. In this respect, they would be considered very accurate at predicting human adverse health effects in industrial exposure situations.

- Occupational limits are determined for a population of workers who are essentially healthy and who fall within a working age group of about 17 to 65 years. These individuals are supposedly in the prime of life, and potentially less susceptible to the effects of hazardous substances than other members of the public. Individuals vary in

sensitivity or susceptibility to hazardous substances; with the elderly and infants in general being more susceptible than healthy workers.

- For most substances, a worker during a normal work schedule (8 hours per day, 5 days per week) receives 40 hours of exposure per week with daily breaks and extended weekend periods in which the body may rid itself of the accumulated substances before elevated levels are reached. For a person living continuously in an environment containing such substances; however, these recovery periods do not exist.

For these reasons, agencies using occupational limits have a policy of adjusting them downward with the use of safety or adjustment factors to derive guidelines for environmental (ambient) settings. The occupational limits are considered surrogates for benchmark values for ambient exposures only because they tend to be based upon a large body of toxicological, epidemiological, and/or clinical evidence pertaining to human exposure (albeit in the workplace). Uncertainty exists in terms of whether too little (or too much) safety is inherent in ambient air guidelines developed from occupational limits.

Table 11 Summary of Ambient Air Quality Objectives or Guidelines for Formaldehyde

		Value [$\mu g\ m^{-3}$] Averaging Time:		
Agency*	**Guideline Title**	**1-hour**	**24-hour**	**Annual**
Alberta Environment	Ambient air quality objective	65		
British Columbia Environment	Air Quality Objective			
	action level	60		
	episode level	370		
Manitoba Conservation	Ambient air quality criteria	60		
Ontario	½-hour standard	65 (30-min.)		
	24-hour standard		65	
US ATSDR	Inhalation MRL		50 (14-d)	10 (>365 d)
US EPA	Risk specific Concentration			0.8[1]
Arizona DEQ	Ambient air quality guideline	20	12	0.08
California EPA	Reference exposure level	94		3
Indiana DEM	No guideline exists			
Louisiana DEQ	Ambient air standard			7.3
Massachusetts DEP	Threshold effects exposure limit		0.33	
	Allowable ambient limit			0.08
Michigan DEQ	Initial threshold screening level			0.08
	Secondary risk screening level			0.8
Minnesota DOH	Health risk value	94		0.8
New Hampshire DES	Ambient air limit		1.3	0.88
North Carolina ENR	Acceptable ambient level	150		
Ohio EPA	No guideline exists			
Oklahoma DEQ	Maximum acceptable ambient concentration		12	
Pennsylvania DEP	No guideline exists			
Rhode Island DEM	Acceptable ambient level	50	40	0.08
Texas CEQ	Effects screening level	15		1.5
Vermont ANR	Hazardous ambient air standard			0.08
Washington DOE	Acceptable source impact level			0.077
Wisconsin DNR	No guideline exists			
New Zealand MOE	Health-based guideline value	100 (30-min)		15
The Netherlands (RIVM)	No guideline exists			
World Health Organization	Reference exposure level	100 (30-min)		

* See Appendix B for full name of agency names that have been abbreviated.

[1] RsC shown here to illustrate exposure concentration in air associated with a 1 in 100,000 lifetime cancer risk (risk criteria used in Alberta).

9.0 REFERENCES

Achatz, S., G. Lorinci, N. Hertkorn, I. Gebelgi and A. Kettrup. 1999. Disturbance of the Determination of Aldehydes and Ketones. *Fres. J. Anal. Chem.* 364: 141-146.

Agency for Toxic Substances and Disease Registry (ATSDR). 1999. *Toxicological Profile for Formaldehyde*. ATSDR, Public Health Service, US Department of Health and Human Services. Atlanta, GA. July 1999. 468 pp. (available at http://www.atsdr.cdc.gov/toxprofiles/tp111.html).

Agency for Toxic Substances and Disease Registry (ATSDR). 2005. Minimal Risk Levels (MRLs) for Hazardous Substances. ATSDR, Public Health Service, US Department of Health and Human Services. Atlanta, GA. http://www.atsdr.cdc.gov/mrls.html (accessed 15 October 2005).

Alberta Environment. 2005. Alberta Ambient Air Quality Objectives. Alberta Environment, Environmental Policy Branch, Edmonton, AB. April 2005. 4 pp. http://www3.gov.ab.ca/env/protenf/approvals/factsheets/ABAmbientAirQuality.pdf (accessed 15 October 2005).

Aragon, P., J. Atienza and M.D. Climent. 2000. Analysis of Organic Compounds in Air: A Review. *Critical Reviews in Analytical Chemistry* 30(2&3): 121-151.

Arizona Department of Environmental Quality (DEQ). 2005. Arizona Ambient Air Quality Guidelines. Arizona DEQ, Air Quality Division, Phoenix, AZ. 10 pp.http://www.azdeq.gov/environ/air/index.html (accessed 15 October 2005).

Arizona Department of Health Services (DHS). 1999. 1999 Update – Arizona Ambient Air Quality Guidelines (AAAQGs). Report prepared for Arizona Department of Environmental Quality, Air Programs Division. Arizona DHS, Office of Environmental Health, Phoenix, AZ. 11 May 1999. 20 pp.

Atkinson, R. 1995. Gas Phase Tropospheric Chemistry of Organic Compounds, in *Volatile Organic Compounds in the Atmosphere*, Hester, R.E. and Harrison, R.M. (eds.), Royal Society of Chemistry, Cambridge, UK.

British Columbia Ministry of Environment (MOE). 2005. Air Quality Objectives and Standards, Air Quality Objectives for British Columbia and Canada. British Columbia MOE, Victoria, BC http://wlapwww.gov.bc.ca/air/airquality/pdfs/aqotable.pdf (accessed 15 October 2005).

Brown, R.H. and M.D. Wight. 1994. Diffusive Sampling Using Tube-type Samplers. *Analyst* 119: 75-79.

California Office of Environmental Health Hazard Assessment (OEHHA). 1999. Acute Toxicity Summary: Formaldehyde. Cal OEHHA, Sacramento, CA. 8 pp. http://www.oehha.ca.gov/air/acute_rels/allAcRELs.html (accessed 15 October 2005).

California Office of Environmental Health Hazard Assessment (OEHHA). 2003. Chronic Toxicity Summary: Fluorides including Formaldehyde. Cal OEHHA, Sacramento, CA. 16 pp. http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html (accessed 15 October 2005).

Cardenas, L.M., D.J. Brassinton, B.J. Allen, H. Coe, B. Alicke, U. Platt, K.M. Wilson, J.M.C. Plane and S.A. Penkett. 2000. Intercomparison of Formaldehyde Measurements in Clean and Polluted Atmospheres. *Journal of Atmospheric Chemistry* 37: 53-80.

Chen, I., Chan, S.Y., Cossins, E.A., 1997. Distribution of Folate Derivatives and Enzymes for synthesis of 10-Formyltetrahydrofolate in Cytosolic and Mitochondrial Fractions of Pea Leaves. *Plant Physiol.* 115:299-309.

Cossins, E.A., 1987. Folate biochemistry and metabolism of one carbon units. In: Stump P.K. Conn E.E. (eds). *The Biochemistry of Plants* Vol. 11.

Environment Canada (EC). 2005. *National Pollutant Release Inventory 2003 Database (Access Format)*, (available at http://www.ec.gc.ca/pdb/npri/npri_dat_rep_e.cfm#highlights).

EC. 2004. *2003 Guide for Reporting to the National Pollutant Release Inventory, Canadian Environmental Protection Act, 1999*, Environment Canada (avail. at http://www.ec.gc.ca/pdb/npri/).

EC. 2001. *Priority Substances List Assessment Report: Formaldehyde*. Canadian Environmental Protection Act, Environment Canada/Health Canada, 111 p.

Fried, A., Y.N. Lee, G. Frost, B. Wert, B. Henry, J.R. Drummond, G. Hubler and T. Jobson. 2002. Airborne Formaldehyde Measurements over the North Atlantic during the 1997 NARE Campaign: Instrument Comparisons and Distributions. *Journal of Geophysical Research* 107(D4): 1-21.

Friedfeld, S. M. Fraser, D. Lancaster, D. Leleux, D. Rehle and F. Tittel. 2000. Field Intercomparison of a Novel Optical Sensor for Formaldehyde Quantification. *Geophysical Research Letters* 27(14): 2093-2096.

Fung, Y.S. and Y. Long. 2001. Determination of Carbonyl Compounds in Air by Electrochromatography. *Electrophoresis* 22: 2270-2277.

Gienium Publishing Corporation (Gienium). 1999. *Gienium's Handbook of Safety, Health and Environmental Data for Common Hazardous Substances*, McGraw Hill, New York, New York.

Gerberich, H.R., Seaman, G. C. 2004. Formaldehyde. In *Kirk-Othmer Encyclopedia of Chemical Technology (On-line Edition)*, John Wiley & Sons, Inc.

Giese, M., Bauer-Doranth, U., Langebartels, C., and Sandermann, H. Jr.(1994) Detoxification of Formaldehyde by the Spider Plant (Chlorophytum comosum L.) and by Soybean (Glycine max L.) Cell-Suspension Cultures. Plant Physiol. 104: 1301–1309

Grosjean, E., P.G. Green, D. Grosjean. 1999. Liquid Chromatography Analysis of Carbonyl (2,4-Dinitrophenyl) Hydrazones with Detection by Diode Array Ultraviolet Spectroscopy and by Atmospheric Pressure Negative Chemical Ionization Mass Spectrometry. *Anal. Chem.* 71: 1851-1861.

Haagen-Smit, A.J., Darley, E.E., Zaitlin, M., Hull, H., Noble, W.M., 1952. Investigation on injury to plants from air pollution in the Los Angeles area. *Plant Physiol.*27:18–34.

Hak, C., I. Pundt, S. Trick, C. Kern, U. Platt, J. Dommen, C. Ordonez, A.S.H. Prevot, W. Junkermann, C. Astorga-Llorens. B.R. Larsen, J. Mellqvist, A. Strandberg, Y. Yu, B. Galle, J. Kleffmann, J.C. Lorzer, G.O. Braathen and R. Volkamer. 2005. Intercomparison of Four Different In-Situ Techniques for Ambient Formaldehyde Measurements in Urban Air. *Atmos. Chem. Phys. Discuss.* 5: 2897-2945.

Health Canada. 2005. Proposed residential indoor air quality guideline for formaldehyde. Health Canada, Ottawa, ON. (available at http://canadagazette.gc.ca/partI/2005/20050820/html/notice-e.html#i3).

Howard, P.H. 1989. *Handbook of Environmental Fate and Exposure Data for Organic Chemicals*, Lewis Publishers, Chelsea, MI.

Indiana Department of Environmental Management (DEM). 2002. Office of Air Quality Programs. Indiana DEM, Office of Air Quality. Indianapolis, IN. (available at http://www.in.gov/idem/air/programs/modeling/policy.html).

International Agency for Research on Cancer (IARC). 1995. *Wood Dust and Formaldehyde*, IARC Monograph on the Evaluation of the Carcinogenic Risk of Chemicals to Humans, Vol. 62 (excerpt available at http://monographs.iarc.fr/htdocs/monographs/vol62/formal.html).

IARC (2004). IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Formaldehyde (Group 1). Vol 88 (in preparation). (Posted on-line September 2004 at www-cie.iarc.fr/htdocs/monographs/vol88/formal.html).

IRIS (Summary Information on the Integrated Risk). 1991 (Carcinogenicity Assessment). U.S. Environmental Protection Agency Integrated Risk Information Summary. Formaldehyde (CASRN 50-00-0). (available at www.toxnet.nlm.nih.gov)

Komazaki, Y., Y. Narita and S. Tanaka. 1998. Development of an Automated Measurement System Using a Diffusion Scrubber and High Performance Liquid Chromatography for the Monitoring of Formaldehyde and Acetaldehyde in Automotive Exhaust Gas. *Analyst* 123: 2343-2349.

Larsen, J.C. and Larsen, P.B. 1998. Chemical Carcinogens, in *Air Pollution and Health*, Hester, R.E. and Harrison, R.M. (eds.), Issues in Environmental Science and Technology, Royal Society of Chemistry, Cambridge, UK.

Levart, A. and M. Verber. 2001. Determination of Aldehydes and Ketones in Air Samples Using Cryotrapping Sampling. *Chemosphere* 44: 701-708.

Levin, J.O. and R. Lindahl. 1994. Diffusive Air Sampling of Reactive Compounds – A Review. *Analyst* 119: 79-83.

Lewis, R.J. Sr. 2000. *Sax's Dangerous Properties of Industrial Materials 10th Edtion (On-line Edition)*, John Wiley & Sons, Inc.

Lewis, R.J. Sr. 2002. *Hawley's Condensed Chemical Dictionary 14th Edition (Online Edition)*, John Wiley & Sons, Inc.

Li, Y., J.D. Wang, Z.H. Huang, H.Q. Xu and X.T. Zhou. 2002. Monitoring Leking Gases by OP-FTIR Remote Sensing. *Journal of Environmental Science and Health*A37 (8): 1453-1462.

Li, Y., K.L. Demerjian, M.S. Zahniser, D.D. Nelson, J.B. McManus and S.C. Herndon. 2004. Measurement of Formaldehyde, Nitrogen Dioxide and Sulphur Dioxid at Whiteface Mountain using a Dual Tunable Diode Laser System. *Journal of Geophysical Research* 109: 1-11.

Lide, D.R. (ed.). 2004. *CRC Handbook of Chemistry and Physics, 85th Edition*. CRC Press, Boca Raton, FL.

Louisiana Department of Environmental Quality (DEQ). 2003. Louisiana Administrative Code (LAC). Title 33 Environmental Quality, Part III Air, Chapter 51. Comprehnsive Toxic Air Pollutant Emission Control Program. Louisiana Department of Environmental Quality. Baton Rouge, LA. (available at http://www.state.la.us/osr/lac/lac3.htm).

Maeda, Y., X. Hu, S. Itou, M. Kitano, N. Takenaka, H. Bandow and M. Mnemori. 1994. Continuous Determination of Gaseous Formaldehyde by a Chemiluminescence Method. *Analyst* 119: 2237-2240.

Maeda, Y., X. Hu, S. Itou, M. Kitano, N. Takenaka, H. Bandow and M. Mnemori. 1994. Continuous Determination of Gaseous Formaldehyde by a Chemiluminescence Method. *Analyst* 119: 2237-2240.

Manitoba Conservation. 2005. Objectives and Guidelines for Various Air Pollutants: Ambient Air Quality Criteria (updated July, 2005). Manitoba Conservation, Air Quality Section, Winnipeg, MB. (available at http://www.gov.mb.ca/conservation/airquality/aq-criteria/ambientair_e.html).

Masaru, N., Syozo, F., Saburo, K., 1976. Effects of exposure to various injurious gases on germination of lily pollen. *Environmental Pollution*, 11:181–188.

Massachusetts Department of Environmental Protection (DEP). 1990. Chemical Health Effects Assessment Methodology & Method to Derive Allowable Ambient Limits (CHEM/AAL).Massachusetts DEP, Boston, MA. February 1990. (available at http://www.mass.gov/dep/air/laws/policies.htm).

Massachusetts Department of Environmental Protection (DEP). 1995. Revised air guidelines [updated list of 24-hour average Threshold Effects Exposure Limit (TEL) values and annual average Allowable Ambient Limit (AAL) values]. Memorandum. Massachusetts DEP, Boston, MA. 6 December 1995. (available at http://www.mass.gov/dep/air/aallist.pdf).

Michigan Department of Environmental Quality (DEQ). 1998. Michigan Administrative Code (MAC). Air Pollution Control Rules. Part 2 Air Use Approval, R 336.1201 - 336.1299. Air Quality Division, Department of Environmental Quality. Lansing, MI. (available at http://www.state.mi.us/orr/emi/admincode.asp?admincode=Department&Dpt=EQ).

Michigan Department of Environmental Quality (DEQ). 2005. Initial Threshold Screening Level (ITSL) / Initial Risk Screening Level (IRSL) Glossary. Michigan DEQ, Air Quality division, Lansing, MI. (available at http://www.michigan.gov/deq/0,1607,7-135-3310_4105-11754--,00.html).

Minnesota Department of Health (DOH). 2005. Health Risk Values for Air. Minnesota DOH, St. Paul, MN. (available at http://www.health.state.mn.us/divs/eh/air/hrvtable.htm#chronic).

Muir P.S., Shirazi, A.M., 1997. Effects of Formaldehyde-Enriched Mists on Psuedotsuga menziiesii (Mirbel) franco and Lobararia pulmonaria (L.) Hoffm. *Environ. Pollut.* 94:227-234.

Muntuta-Kinyanta, C. and J.K. Hardy. 1991. Permeation-Solid Absorbent and GC Analysis of Formaldehyde. *Talanta* 38(12): 1381-1386.

Mutters, R.G., Madore, M., Bytnerowicz, A., 1993. Formaldehyde exposure affects growth and metabolism of common bean. *Journal of the Air and Waste Management Association* 43:113–116.

New Hampshire Department of Environmental Services (DES). 2005. New Hampshire Administrative Rule. Chapter Env-A 1400. Regulated Toxic Air Pollutants. New Hampshire Department of Environmental Services. Concord, NH. http://www.des.state.nh.us/rules/env-a1400.pdf (accessed 15 October 2005).

New Jersey Department of Environmental Protection (DEP). 1994. Technical Manual 1003. Guidance on Preparing a Risk Assessment for Air Contaminant Emissions. Air Quality Permitting Program, Bureau of Air Quality Evaluation, New Jersey DEP, Trenton, NJ. Revised December 1994. http://www.state.nj.us/dep/aqpp/downloads/techman/1003.pdf (accessed 15 October 2005).

New Jersey Department of Environmental Protection (DEP). 2005. New Jersey Administrative Code (NJAC). Title 7, Chapter 27, Subchapter 8. Permits and Certificates for Minor Facilities (and Major Facilities without an Operating Permit). New Jersey Department of Environmental Protection. Trenton, NJ. http://www.state.nj.us/dep/aqm/rules.htm (accessed 15 October 2005).

New Zealand Ministry for the Environment and Ministry of Health (New Zealand). 2002. Ambient Air Quality Guidelines 2002 Update. Prepared by the Ministry for the Environment and the Ministry of Health. Wellington, New Zealand. May 2002. 58 pp. www.mfe.govt.nz (accessed 3 February 2006).

NIOSH (National Institute for Occupational Safety and Health). 2003. *NIOSH Manual of Sampling and Analytical Methods – 4th Edition, Method 2016, Issue 2*. US Department of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Physical Sciences and Engineering, Cincinnati, OH, 2003.

NIOSH. 1994a. *NIOSH Manual of Sampling and Analytical Methods – 4th Edition, Method 2541, Issue 2*. US Department of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Physical Sciences and Engineering, Cincinnati, OH, 1994.

NIOSH. 1994b. *NIOSH Manual of Sampling and Analytical Methods – 4th Edition, Method 3500, Issue 2*. US Department of Health, Education, and Welfare, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, Division of Physical Sciences and Engineering, Cincinnati, OH, 1994.

North Carolina Department of Environment and Natural Resources. 2005. North Carolina Administrative Code (NCAC). North Carolina Air Quality Rules 15A NCAC 2D.1100 – Air Pollution Control Requirements (Control of Toxic Air Pollutants). North Carolina Department of Environment and Natural Resources, Raleigh, NC. (available at http://reports.oah.state.nc.us/ncac.asp).

O'Neil, M. (Ed.). 2001. *The Merck Index, 13th Edition*, Merck Research Laboratories, Division of Merck & Co., Inc. Whitehouse Station, N.J.

OECD SIDS (Organisation for Economic Co-operation and Development Screening Information Datasheet). 2002. Formaldehyde CAS NO. 50-00-0. SIDS Initial Assessment Report (SIAR) for SIAM 14. Paris, France, March 2002. UNEP Publications. (available at www.chem.unep.ch/irptc/sids/OECDSIDS/FORMALDEHYDE.pdf)

Ohio Environmental Protection Agency (EPA). 2005. Air Toxics Policy – Option A: Review of New Sources of Toxic Emissions. Air Toxics Unit, Division of Air Pollution Control, Ohio EPA. Columbus, OH. 11 pp. (available at http://www.epa.state.oh.us/dapc/atu/atu.html).

Ohio Environmental Protection Agency (Ohio EPA). 1994. Review of New Sources of Air Toxic Emissions. Proposed for Public Comment. Division of Air Pollution Control, Ohio EPA. Columbus, OH. January 1994. 31 pp. (available at http://www.epa.state.oh.us/dapc/atu/atu.html).

Oklahoma Department of Environmental Quality (DEQ). 2002. Air Toxics Partial Listing [maximum acceptable ambient concentrations (MAAC) for air toxics]. Oklahoma City, OK. (available at http://www.deq.state.ok.us/AQDNew/toxics/listings/pollutant_query_1.html).

Oklahoma Department of Environmental Quality (DEQ). 2005. Oklahoma Administrative Code (OAC). Title 252. Chapter 100. Air Pollution Control. 100:252-41 - Control of Emission of Hazardous and Toxic Air Contaminants. Oklahoma DEQ, Oklahoma City, OK. (available at http://www.sos.state.ok.us/oar/oar_welcome.htm).

Ontario Ministry of the Environment (OME). 2005. Summary of O. Reg. 419/05 Standards and Point of Impingement Guidelines and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of the Environment, Toronto, ON. December 2005. 16 pp. http://www.ene.gov.on.ca/envision/air/airquality_standards.htm (accessed 3 February 2006).

OSHA (Occupational Safety and Health Administration). 1989. *OSHA Sampling and Analytical Methods. Acrolein and/or Formaldehyde Method 52.* Organic Methods Evaluation Branch, Occupational Safety and Health Administration, US Department of Labor, OSHA Salt Lake Technical Center, Salt Lake City, UT. June 1989.

OSHA. 1990. *OSHA Sampling and Analytical Methods. Formaldehyde in Workplace Atmospheres (3M Model 3721 Monitor) Method ID-205.* Organic Methods Evaluation Branch, Occupational Safety and Health Administration, US Department of Labor, OSHA Salt Lake Technical Center, Salt Lake City, UT. December 1990.

Olson, R. and P. Fellin. 1988. A Review of Techniques for Measurement of Airborne Aldehydes. *The Science of the Total Environment* 77: 95-131.

Pennsylvania Department of Environmental Protection (DEP). 2005. Pennsylvania State Code, Article III Air Resources, Section 131.1. Ambient Air Quality Standards. Pennsylvania DEP, Bureau of Air Quality, Harrisburg, PA (available at http://www.pacode.com/secure/data/025/article3/chapter131_toc.html).

Plog, B.A., J. Niland, P.J. Quinlan. (eds.) 1996. *Fundamentals of Industrial Hygiene 4th Ed.* National Safety Council. Itasca, Il. pp1011.

Possanzini, M., V. Di Pali, Petricca, M. Fratarcangeli and R.D. Brocco. 1996. Measurements of Lower Carbonyls in Rome Ambient Air. *Atmos. Environ.* 30: 3757-3764.

Pretto, A., M.R. Milani and A.A. Cardoso. 2000. Colorimetric Determination of Formaldehyde in Air Using a Hanging Drop of Chromotropic Acid. *J. Environ. Monit.* 2: 566-570.

Reuss, G., Dstledorf, W., Gamer, A.O., Hilt. A. 2002. Formaldehyde. In *Ullman's Encyclopedia of Industrial Chemistry (On-line Edition)*, Wiley-VCH Verlag GmbH & Co. KGaA.

Rhode Island Department of Environmental Management. 2004. Air Pollution Control Regulation #22, Air Toxics. Division of Air and Hazardous Materials, Rhode Island Department of Environmental Management. Providence, RI. Amended 27 April 2004. (available at http://www.state.ri.us/dem/pubs/regs/index.htm#Air).

Sakuragawa, A., T. Yoneno, K. Inoue, and T. Okutani. 1999. Trace Analysis of Carbonyl Compounds by Liquid Chromatography-Mass Spectrometry after Collection as 2,4-Dinitrophenylhydrazine Derivatives. *J. Chromatogr. A.* 844: 403-408.

Sardi, E., Tyihak, E., 1994. Si mple Determination of Formaldehyde in Dimedone Adduct Form in Biological Samples by High Performance Liquid Chromotography. *Biomed. Chromotograph.* 8:313-314.

Sandau, F., W. Dott and J. Hollender. 2001. Sensitive Indoor Air Monitoring of Formaldehyde and Other Carbonyl Compounds using the 2,4-Dinotrophenylhydrazine. *Int. J. Hyg. Environ. Health* 203: 275-279.

Sweet, C., .T. Pisano and D.R. Fitz. 2003. TunableDiode Laser Absorption Spectrometer Meaurments of Ambient Nitrogen Dioxide, Nitric Acid, Formaldehyde, and Hydrogen Peroxide in Parlier, California. *Atmospheric Environment* 37: 1583-1591.

Shirazi, A.M., and Muir, P.S., 1998. In Vitro effect of Formaldehyde on Douglas fir Pollen. *PlantCell Environ.* 21:341-346.

Syracuse Research Corporation (SRC). 2005. Environmental Fate Database (available at http://www.syrres.com/esc/efdb.htm).

Texas Commission on Environmental Quality (CEQ). 2003. Effects Screening Levels. TCEQ Toxicology Section, Austin, TX. (available at http://www.tceq.state.tx.us/implementation/tox/esl/list_main.html).

The Netherlands National Institute of Public Health and the Environment (RIVM). 2001. Re-evaluation of human-toxicological maximum permissible risk levels. RIVN Report 711701 025. RIVN, Bilthoven, The Netherlands. March 2001. 297 pp.

Thomas, C.L.P., C.D. McGill and R. Towill. 1997. Determination of Formaldehyde by Conversion to Hexahydrooxazolo[3,4-a]pyridine in a Denuder Tube With Recovery by Thermal Desorption, and Analysis by Gas Chromatography-Mass Spectrometry. *Analyst* 121: 1471-1476.

Tuazon, E.C., R.A. Graham, A.M. Winer, R.R. Easton and J.N. Pitts Jr. 1978. A Kilometre Pathlength Fourier-Transform Infrared System for the Study of Trace Pollutants in Ambient Air and Synthetic Atmospheres. *Atmos. Environ.* 12: 865-875.

Uchiyama, S. and S. Hasegawa. 1999. A Reactive and Sensitive Diffusion Sampler for the Determination of Aldehydes and Ketones in Ambient Air. *Atmos. Environ.* 33: 1999-2005.

US Environmental Protection Agency. 2005. Integrated Risk Information System. http://www.epa.gov/iris/ (accessed 15 October 2005).

US EPA (United States Environmental Protection Agency). 1999. Compendium of Methods for the Determination of Toxic Organic Compounds in Ambient Air – 2nd Edition. US Environmental Protection Agency, Office Research and Development, National Risk Management Research Laboratory, Centre for Environmental Research Information. Cincinnati, Ohio. January 1999. EPA/625/R-96/010b.

Vairavamurthy, A., Roberts J.M. and L. Newman. 1992. Methods for Determination of Low Molecular Weight: Carbonyl Compounds in the Atmosphere: A Review. *Atmos Environ* 26A: 1965-1993.

Vermont Agency of Natural Resources (ANR). 2001. Air Pollution Control Regulations. State of Vermont Agency of Natural Resources, Air Pollution Control Division, Waterbury, VT. http://www.anr.state.vt.us/air/AirToxics/docs/apcregs.pdf (accessed 15 October 2005).

Washington State Department of Ecology (DOE). 2005. Washington Administrative Code (WAC). Chapter 173-460 WAC. Controls For New Sources Of Toxic Air Pollutants. Washington State DOE, Olympia, WA. http://www.leg.wa.gov/wac/ (accessed 15 October 2005).

Wisconsin Department of Natural Resources (DNR). 2005. Wisconsin Administrative Code (WAC). Air Pollution Control Rules. Chapter NR 445. Control of Hazardous Pollutants. Wisconsin DNR, Madison WI. http://www.legis.state.wi.us/rsb/code/nr/nr445.pdf (accessed 15 October 2005).

World Health Organization (WHO). 2002. Formaldehyde. Concise International Chemical Assessment Document Vol 40. World Health Organization. Geneva, 2002. (available at www.inchem.org/documents/WHO/WHO/cicad40.htm)

WHO. 2000. *Air Quality Guidelines for Europe*, 2nd Edition. WHO Regional Publications, European Series, No. 91. WHO Regional Office for Europe, Copenhagen. 273 pp.

WHO. 1989. *Environmental Health Criteria 89: Formaldehyde*. International Programme on Chemical Safety, World Health Organization, Geneva, Switzerland (available at http://www.inchem.org/documents/ehc/ehc/ehc89.htm).

Zhang, J., L. Zhang, Z. Fan and V. Ilacqua. 2000. Development of the Personal Aldehydes and Ketones Sampler Based upon DNSH Derivation on Solid Sorbent. *Environ. Sci. Technol.* 34: 2601-2607.

Zhou, X. and K. Mopper. 1990. Measurement of Sub-Parts-per-Billion Levels of Carbonyl Compounds in Marine Air by a Simple Cartridge Trapping Procedure Followed by Liquid Chromatography. *Environ. Sci. Technol.* 24: 1482-1485.

APPENDIX A
Formaldehyde Emissions
2003 NPRI Database

Table A-1 Total Formaldehyde Emissions According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by province and by total emissions)

NPRI ID	Company	City	Province	Formaldehyde Emissions (in tones)			
				Air	Water	Land	Total
4880	Ainsworth Lumber Co. Ltd.	Grande Prairie	AB	208.69	0	0	208.686
2762	Weyerhaeuser Canada Ltd.	Edson	AB	40.744	0	0	40.744
2764	Weyerhaeuser Company Limited	Slave Lake	AB	18.5	0	0	18.5
1251	Owens-Corning Canada Inc.	Edmonton	AB	17.718	0	0	17.718
0001	Alberta Pacific Forest Industries	Boyle	AB	10.86	0.14	0	11
3941	SOLEX Gas Processing Corp	Didsbury	AB	10.36	0	0	10.36
5285	Apache Canada Limited	Zama City	AB	10.1	0	0	10.1
4830	West Fraser Mills	Blue Ridge	AB	10	0	0	10
6647	Albian Sands Energy Inc.	Ft. McMurray	AB	7.368	0	0	7.368
15437	ATCO Gas	Carbon	AB	7.254	0	0	7.254
3821	Canadian Fertilizers limited	Medicine Hat	AB	7.192	0	0	7.192
1902	Nexen Canada Ltd.	Balzac	AB	4.25	0	0	4.25
0011	Borden Chemical Canada, Inc.	Edmonton	AB	3.829	0	0	3.829
6517	Footner Forest Products Ltd.	High Level	AB	1.15	0	0	1.15
3269	Agrium Inc	Calgary	AB	0.8	0	0	0.8
2963	Shell Chemicals Canada Ltd.	Fort Saskatchewan	AB	0.626	0	0	0.626
5351	Baker Petrolite Corporation	Calgary	AB	0	0	0	0.493
2316	Dow Chemical Canada Inc.	Red Deer	AB	0.486	0	0	0.486
1162	Celanese Canada Inc.	Edmonton	AB	0.345	0	0.001	0.346
0280	Dow Chemical Canada Incorporated	Fort Saskatchewan	AB	0.056	0	0	0.056
6512	Norwood Foundry Ltd.	Nisku	AB	0.011	0	0	0.011
7904	Target Products Ltd	Calgary	AB	0	0	0	0.002
7905	Target Products Ltd	Mornville	AB	0	0	0	0.002
0853	Marsulex Inc.	Fort Saskatchewan	AB	0.002	0	0	0.002
2291	Brenntag Canada Inc. (AS65)	Calgary	AB	0	0	0	0.001

Table A-1 Total Formaldehyde Emissions According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by province and by total emissions) (continued)

NPRI ID	Company	City	Province	Formaldehyde Emissions (in tones)			
				Air	Water	Land	Total
2340	Univar Canada Ltd.	Calgary	AB	0.001	0	0	0.001
5108	West Fraser Mills Ltd.	Quesnel	BC	102.6	0	0	102.6
0333	NorskeCanada	Campbell River	BC	63.84	0	0	63.84
5127	Slocan Forest Products Ltd.	Fort Nelson	BC	60.2	0	0	60.2
5191	Ainsworth Lumber Company	100 Mile House	BC	24.593	0	0	24.593
1266	Norske Skog Canada Limited	Crofton	BC	21.4	0.98	0	22.38
0718	Louisiana-Pacific Canada Ltd.	Dawson Creek	BC	21.842	0	0	21.842
0479	Cariboo Pulp and Paper Co.	Quesnel	BC	16.9	2.74	0	19.64
4063	Canfor - Prince George Pulp and Paper Mills	Prince George	BC	8.895	4.044	0	12.939
2924	Weyerhaeuser Company Limited	Kamloops	BC	9.032	3.67	0	12.702
1797	Canadian Forest Products Ltd.	Prince George	BC	8.769	1.874	0	10.643
2872	Western Pulp Limited Partnership	Squamish	BC	6.4	0	0	6.4
7862	Ainsworth Lumber Co. Ltd	Savona	BC	4.225	0	0	4.225
5128	Canadian Forest Products Ltd.	New Westminster	BC	3.5	0	0	3.5
5126	Roxul West Inc.	Grand Forks	BC	1.245	0	0	1.245
1693	Dynea Canada Limited	Kamloops	BC	0.578	0	0	0.578
0013	Borden Chemical Canada Inc.	Vancouver	BC	0	0	0	0.357
7906	Target Products Ltd	Abbotsford	BC	0	0	0	0.002
2515	Simplot Canada Limited	Brandon	MB	0	0	0	0.2
5277	Elias Woodworking and Manufacturing Ltd	Winkler	MB	0	0	0	0.098
6823	American Biaxis Inc.	Winnipeg	MB	0.001	0	0	0.001
5003	Weyerhaeuser Company Limited	Miramichi	NB	117.65	0	0	117.654
4842	Flakeboard Company Limited	St. Stephen	NB	63.4	0	0	63.4
2604	Irving Pulp & Paper Limited / Irving Tissue Company	Saint John	NB	5.655	16.39	0	22.045

Table A-1 Total Formaldehyde Emissions According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by province and by total emissions) (continued)

NPRI ID	Company	City	Province	Formaldehyde Emissions (in tones)			
				Air	Water	Land	Total
1617	UPM-Kymmene Miramichi Incorporated	Miramichi	NB	5.45	0.92	0	6.37
5014	Brooklyn Power Corporation	Brooklyn, Queens County	NS	8.84	0	0	8.84
6008	Dalhousie University	Halifax	NS	0	0	0	0.287
5609	TEMPLE-INLAND FOREST PRODUCTS	PEMBROKE	ON	116.3	0	0	116.303
5861	Grant Forest Products Inc.	Timmins	ON	59.49	0	0	59.49
5885	G-P FLAKEBOARD	Sault Ste Marie	ON	45.543	0	0	45.543
1687	Dynea Canada Ltd.	North Bay	ON	26.62	0	0	26.62
1596	Tembec Industries Inc.	Smooth Rock Falls	ON	25	0	0	25
1245	Owens-Corning Canada	Scarborough	ON	20.293	0	0	20.293
4892	Weyerhaeuser Company Limited	Wawa	ON	16.883	0	0	16.883
7170	Ainsworth Engineered Corp.	Barwick	ON	16.1	0	0	16.1
0177	Coveright Surfaces Canada Inc.	Cobourg	ON	15.682	0	0	15.682
1684	Dynea Canada Ltd	Thunder Bay	ON	14.662	0	0	14.662
3870	General Motors of Canada Limited	Oshawa	ON	13.839	0	0	13.839
1882	Fibrex Insulations Inc.	Sarnia	ON	13.748	0	0	13.748
3893	General Motors of Canada Limited	Oshawa	ON	12.746	0	0	12.746
3071	Suncor Energy Products Inc.	Sarnia	ON	11.983	0	0	11.983
1199	PANOLAM INDUSTRIES LTD.	Huntsville	ON	11.3	0	0	11.3
2363	LONGLAC WOOD INDUSTRIES	Longlac	ON	11.288	0	0	11.288
1111	Uniboard Canada Inc.	New Liskeard	ON	9.949	0	0	9.949
2656	Ventra Group	Peterborough	ON	8.59	0	0	8.59
5985	Rieter Automotive Mastico Limited	Tillsonburg	ON	7.911	0	0	7.911
4559	Grant Forest Products Inc.	Englehart	ON	7.477	0.09	0	7.567
3013	Norampac Inc.	Red Rock	ON	6.272	0	0	6.272
3201	3M Canada Company (Perth)	Perth	ON	5.618	0	0	5.618

Table A-1 Total Formaldehyde Emissions According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by province and by total emissions) (continued)

NPRI ID	Company	City	Province	Formaldehyde Emissions (in tones)			
				Air	Water	Land	Total
3949	Karmax Heavy Stamping	Milton	ON	4.936	0	0	4.936
5689	Guardian Building Products Distribution Canada Inc.	Erin	ON	4.71	0	0	4.71
0462	Marathon Pulp Inc.	Marathon	ON	4.3	0	0	4.3
5687	ROXUL INC	Milton	ON	2.85	0	0	2.85
1857	Ottawa Fibre Inc.	Ottawa	ON	2.43	0	0	2.43
3182	Saint-Gobain	Plattsville	ON	1.434	0	0	1.434
3855	Stelco Inc.	Haldimand County	ON	1.075	0	0	1.075
0151	Canada Alloy Company	Kitchener	ON	0.85	0	0	0.85
0009	Borden Chemical Canada, Inc.	North Bay	ON	0	0	0	0.511
1207	DUPONT CANADA INC.	Maitland	ON	0.5	0	0	0.5
2322	Crompton Co.	Elmira	ON	0.339	0	0	0.339
5954	Greif Bros. Canada	Stoney Creek	ON	0	0	0	0.283
5625	CLEAN HARBORS INC.	Thorold	ON	0.268	0	0	0.268
7109	Iogen Corporation	Ottawa	ON	0.236	0	0	0.236
5955	Greif	Oakville	ON	0	0	0	0.207
10745	Leda Furniture Ltd.	Toronto	ON	0	0	0	0.2
4849	Tarxien Automotive Products Ltd	Concord	ON	0.2	0	0	0.2
2233	Terra International Canada Ltd	Courtright	ON	0	0	0	0.151
10765	MTI	Mississauga	ON	0	0	0	0.132
3198	3M CANADA	London	ON	0.114	0	0	0.114
5915	ABCgroup Inc.	Toronto	ON	0	0	0	0.09
3433	ISP (Canada) Inc.	Toronto	ON	0.054	0	0	0.054
7086	Hartford Fibres Limited	Kingston	ON	0	0	0	0.051
1785	NOVA Chemicals (Canada) Ltd.	Sarnia	ON	0.044	0	0	0.044

Table A-1 Total Formaldehyde Emissions According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by province and by total emissions) (continued)

NPRI ID	Company	City	Province	Formaldehyde Emissions (in tones)			
				Air	Water	Land	Total
1547	Metal Technologies	Woodstock	ON	0.014	0	0	0.014
10611	Deslaurier Custom Cabinets	Renfrew	ON	0.014	0	0	0.014
7004	Airboss of America Corporation	Kitchener	ON	0.013	0	0	0.013
0500	Colgate-Palmolive Canada Inc.	Toronto	ON	0	0	0	0.01
0031	BASF Canada	Windsor	ON	0.009	0	0	0.009
4175	Schenectady Canada Ltd.	Toronto	ON	0.007	0	0	0.007
0222	Cytec Canada Inc.	Niagara Falls	ON	0.004	0	0	0.004
7096	Archie McCoy Hamilton Ltd.	Troy	ON	0	0	0	0.002
10468	Assured Packaging Inc.	Mississauga	ON	0	0	0	0.002
1953	PPG Canada Inc.	Mississauga	ON	0	0	0	0.001
0249	Ashland Canada Corporation	Ajax	ON	0.001	0	0	0.001
5526	Société en commandite Tafisa Canada	Lac-Mégantic	QC	84.1	0.165	0	84.265
2989	Uniboard Canada inc.	Sayabec	QC	79.86	0	0	79.86
0758	Uniboard Canada Inc.	Mont-Laurier	QC	68.04	0	0	68.04
5518	Louisiana Pacific Canada Ltd	Bois-Franc	QC	66.006	0	0	66.006
1745	Industries Norbord Inc.	Val d'Or	QC	57.25	0	0	57.25
4060	Uniboard Canada Inc.	Val-d'Or	QC	55.02	0	0	55.02
5442	Uniboard Canada Inc.	La Baie	QC	52.78	0	0	52.78
1681	Dynea Canada Ltée	Ste Therese	QC	18.343	0	0	18.343
3242	SFK Pâte S.E.N.C	St-Félicien	QC	4.831	13.27	0	18.099
1748	Industries Norbord Inc.	La Sarre	QC	15.77	0	0	15.77
2948	Tembec Inc	Témiscaming	QC	2.988	12.67	0	15.662
5516	Louisiana Pacific Canada Ltd	Saint-Michel-des-Saints	QC	14.584	0	0	14.584
5515	KRUGER	Trois-Rivières	QC	13.492	0	0	13.492
0279	Domtar Inc.	Lebel-sur-Quévillon	QC	4.726	7.201	0	11.927

Table A-1 Total Formaldehyde Emissions According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by province and by total emissions) (continued)

NPRI ID	Company	City	Province	Formaldehyde Emissions (in tones)			
				Air	Water	Land	Total
4068	Papier Stadacona Ltee	Québec	QC	1.615	9.027	0	10.642
4362	Isolation Manson inc.	Brossard	QC	10.6	0	0	10.6
1858	Owens-Corning Canada Inc.	Candiac	QC	7.2	0	0	7.2
0929	BOWATER	Gatineau	QC	2	2.71	0	4.71
6441	Uniboard Canada Inc	Laval	QC	3.6	0	0.58	4.18
2001	La Compagnie Abitibi-Consolidated du Canada	Baie-Comeau	QC	2.597	1.5	0	4.097
3381	Uniboard Canada Inc.	Val-d'Or	QC	3.87	0	0	3.87
4386	Tembec Inc.	St-Georges-de-Champlain	QC	2.9	0	0	2.9
0007	Borden Chimie Canada, Inc.	Laval	QC	1.554	0	0	1.554
4802	Produits Chimiques Handy Ltee	Candiac	QC	1.4	0	0	1.4
5429	Tembec Inc.	Longueuil	QC	1.3	0	0	1.3
5430	3io corp	Riviere Trois-Pistoles	QC	0.67	0	0	0.67
1648	Solutia Canada inc.	LaSalle	QC	0.309	0	0	0.309
6234	Fournitures Funéraires Victoriaville inc.	Victoriaville	QC	0.169	0	0	0.169
5449	Clean Harbors	Mercier	QC	0.162	0	0	0.162
5455	Services environnementaux Clean Harbors Québec, Inc	Thurso	QC	0.154	0	0	0.154
3095	BAKOR INC.	LACHINE	QC	0.15	0	0	0.15
4407	Arborite,division de ITW Canada	LaSalle	QC	0.147	0	0	0.147
5556	Borden Chimie Canada, Inc.	St-Romuald	QC	0.141	0	0	0.141
0330	Ferox Inc / Laques International Inc	Anjou	QC	0.1	0	0	0.1
6289	JB Martin	St-Jean sur Richelieu	QC	0	0	0	0.05
3125	Société Viasystems Canada S.E.N.C.	Kirkland	QC	0.022	0	0	0.022
5454	CleanHarbors environmental services, Inc.	Ste-Catherine	QC	0.02	0	0	0.02
2334	Univar Canada Ltd.	Valleyfield	QC	0.007	0	0	0.007
20040	Meadow Lake OSB Limited Partnership	Meadow Lake	SK	16.367	0	0	16.367
2077	Saskferco Products Inc.	Belle Plaine	SK	0	0	0	0.017

Table A-2 Formaldehyde Air Emissions in Alberta According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by province and by total emissions)

NPRI ID	Company	City	Province	Formaldehyde Emissions (in tonnes)					
				Stack /Point	Storage /Handling	Fugitive	Spills	Other Non-Point	Total
4880	Ainsworth Lumber Co. Ltd.	Grande Prairie	AB	208.686	0	0	0	0	208.69
2762	Weyerhaeuser Canada Ltd.	Edson	AB	40.744	0	0	0	0	40.744
2764	Weyerhaeuser Company Limited	Slave Lake	AB	18.5	0	0	0	0	18.5
1251	Owens-Corning Canada Inc.	Edmonton	AB	17.7	0.018	0	0	0	17.718
0001	Alberta Pacific Forest Industries	Boyle	AB	10.86	0	0	0	0	10.86
3941	SOLEX Gas Processing Corp	Didsbury	AB	10.36	0	0	0	0	10.36
5285	Apache Canada Limited	Zama City	AB	10.1	0	0	0	0	10.1
4830	West Fraser Mills	Blue Ridge	AB	10	0	0	0	0	10
6647	Albian Sands Energy Inc.	Ft. McMurray	AB	7.368	0	0	0	0	7.368
15437	ATCO Gas	Carbon	AB	7.254	0	0	0	0	7.254
3821	Canadian Fertilizers limited	Medicine Hat	AB	7.096	0	0.075	0	0.021	7.192
1902	Nexen Canada Ltd.	Balzac	AB	4.25	0	0	0	0	4.25
0011	Borden Chemical Canada, Inc.	Edmonton	AB	3.373	0.349	0.107	0	0	3.829
6517	Footner Forest Products Ltd.	High Level	AB	1.15	0	0	0	0	1.15
3269	Agrium Inc	Calgary	AB	0	0	0.8	0	0	0.8
2963	Shell Chemicals Canada Ltd.	Fort Saskatchewan	AB	0.409	0.217	0	0	0	0.626
2316	Dow Chemical Canada Inc.	Red Deer	AB	0.486	0	0	0	0	0.486
1162	Celanese Canada Inc.	Edmonton	AB	0	0.241	0.104	0	0	0.345
0280	Dow Chemical Canada Incorporated	Fort Saskatchewan	AB	0	0	0	0	0.056	0.056
6512	Norwood Foundry Ltd.	Nisku	AB	0	0	0.011	0	0	0.011
0853	Marsulex Inc.	Fort Saskatchewan	AB	0.002	0	0	0	0	0.002
2340	Univar Canada Ltd.	Calgary	AB	0	0.001	0	0	0	0.001
5108	West Fraser Mills Ltd.	Quesnel	BC	102.6	0	0	0	0	102.6
0333	NorskeCanada	Campbell River	BC	61.35	2.49	0	0	0	63.84

Table A-2 Formaldehyde Air Emissions in Alberta According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by province and by total emissions) (continued)

NPRI ID	Company	City	Province	Formaldehyde Emissions (in tonnes)					
				Stack /Point	Storage /Handling	Fugitive	Spills	Other Non-Point	Total
5127	Slocan Forest Products Ltd.	Fort Nelson	BC	60.2	0	0	0	0	60.2
5191	Ainsworth Lumber Company	100 Mile House	BC	24.593	0	0	0	0	24.593
1266	Norske Skog Canada Limited	Crofton	BC	21.4	0	0	0	0	21.4
0718	Louisiana-Pacific Canada Ltd.	Dawson Creek	BC	21.842	0	0	0	0	21.842
0479	Cariboo Pulp and Paper Co.	Quesnel	BC	16.9	0	0	0	0	16.9
4063	Canfor - Prince George Pulp and Paper Mills	Prince George	BC	8.85	0	0.045	0	0	8.895
2924	Weyerhaeuser Company Limited	Kamloops	BC	8.97	0	0.062	0	0	9.032
1797	Canadian Forest Products Ltd.	Prince George	BC	8.71	0	0.059	0	0	8.769
2872	Western Pulp Limited Partnership	Squamish	BC	6.4	0	0	0	0	6.4
7862	Ainsworth Lumber Co. Ltd	Savona	BC	4.025	0	0.2	0	0	4.225
5128	Canadian Forest Products Ltd.	New Westminster	BC	3.5	0	0	0	0	3.5
5126	Roxul West Inc.	Grand Forks	BC	1.245	0	0	0	0	1.245
1693	Dynea Canada Limited	Kamloops	BC	0.157	0.03	0.391	0	0	0.578
6823	American Biaxis Inc.	Winnipeg	MB	0.001	0	0	0	0	0.001
5003	Weyerhaeuser Company Limited	Miramichi	NB	117.654	0	0	0	0	117.65
4842	Flakeboard Company Limited	St. Stephen	NB	63.4	0	0	0	0	63.4
2604	Irving Pulp & Paper Limited / Irving Tissue Company	Saint John	NB	5.655	0	0	0	0	5.655
1617	UPM-Kymmene Miramichi Inc.	Miramichi	NB	5.42	0	0.03	0	0	5.45
5014	Brooklyn Power Corporation	Brooklyn, Queens County	NS	8.84	0	0	0	0	8.84
5609	Temple-Inland Forest Products	Pembroke	ON	116.303	0	0	0	0	116.3
5861	Grant Forest Products Inc.	Timmins	ON	59.49	0	0	0	0	59.49
5885	G-P FLAKEBOARD	Sault Ste Marie	ON	36.726	0	8.817	0	0	45.543

Table A-2 Formaldehyde Air Emissions in Alberta According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by province and by total emissions) (continued)

NPRI ID	Company	City	Province	Formaldehyde Emissions (in tonnes)					
				Stack /Point	Storage /Handling	Fugitive	Spills	Other Non-Point	Total
1687	Dynea Canada Ltd.	North Bay	ON	19.768	0.014	6.8	0	0.038	26.62
1596	Tembec Industries Inc.	Smooth Rock Falls	ON	25	0	0	0	0	25
1245	Owens-Corning Canada	Scarborough	ON	20.195	0.098	0	0	0	20.293
4892	Weyerhaeuser Company Limited	Wawa	ON	16.883	0	0	0	0	16.883
7170	Ainsworth Engineered Corp.	Barwick	ON	16.1	0	0	0	0	16.1
0177	Coveright Surfaces Canada Inc.	Cobourg	ON	15.682	0	0	0	0	15.682
1684	Dynea Canada Ltd	Thunder Bay	ON	10.707	0.391	3.527	0	0.037	14.662
3870	General Motors of Canada Limited	Oshawa	ON	13.839	0	0	0	0	13.839
1882	Fibrex Insulations Inc.	Sarnia	ON	13.748	0	0	0	0	13.748
3893	General Motors of Canada Limited	Oshawa	ON	12.742	0.001	0.003	0	0	12.746
3071	Suncor Energy Products Inc.	Sarnia	ON	11.983	0	0	0	0	11.983
1199	Panolam Industries Ltd.	Huntsville	ON	11.3	0	0	0	0	11.3
2363	Longlac Wood Industries	Longlac	ON	11.288	0	0	0	0	11.288
1111	Uniboard Canada Inc.	New Liskeard	ON	9.949	0	0	0	0	9.949
2656	Ventra Group	Peterborough	ON	8.59	0	0	0	0	8.59
5985	Rieter Automotive Mastico Limited	Tillsonburg	ON	0.014	0	7.897	0	0	7.911
4559	Grant Forest Products Inc.	Englehart	ON	7.477	0	0	0	0	7.477
3013	Norampac Inc.	Red Rock	ON	6.215	0.013	0.044	0	0	6.272
3201	3M Canada Company	Perth	ON	5.618	0	0	0	0	5.618
3949	Karmax Heavy Stamping	Milton	ON	0	0	4.936	0	0	4.936
5689	Guardian Building Products Distribution Canada Inc.	Erin	ON	1.2	3.51	0	0	0	4.71
0462	Marathon Pulp Inc.	Marathon	ON	4.3	0	0	0	0	4.3
5687	ROXUL INC	Milton	ON	2.85	0	0	0	0	2.85
1857	Ottawa Fibre Inc.	Ottawa	ON	2.43	0	0	0	0	2.43

Table A-2 Formaldehyde Air Emissions in Alberta According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by province and by total emissions) (continued)

NPRI ID	Company	City	Province	Formaldehyde Emissions (in tonnes)					
				Stack /Point	Storage /Handling	Fugitive	Spills	Other Non-Point	Total
3182	Saint-Gobain	Plattsville	ON	1.42	0	0.014	0	0	1.434
3855	Stelco Inc.	Haldimand County	ON	1.058	0	0.017	0	0	1.075
0151	Canada Alloy Company	Kitchener	ON	0	0	0	0	0.85	0.85
1207	Dupont Canada Inc.	Maitland	ON	0.28	0	0.22	0	0	0.5
2322	Crompton Co.	Elmira	ON	0.339	0	0	0	0	0.339
5625	Clean Harbors Inc.	Thorold	ON	0.204	0	0.064	0	0	0.268
7109	Iogen Corporation	Ottawa	ON	0.236	0	0	0	0	0.236
4849	Tarxien Automotive Products Ltd	Concord	ON	0.2	0	0	0	0	0.2
3198	3M CANADA	London	ON	0.114	0	0	0	0	0.114
3433	ISP (Canada) Inc.	Toronto	ON	0.054	0	0	0	0	0.054
1785	NOVA Chemicals (Canada) Ltd.	Sarnia	ON	0.044	0	0	0	0	0.044
1547	Metal Technologies	Woodstock	ON	0.014	0	0	0	0	0.014
10611	Deslaurier Custom Cabinets	Renfrew	ON	0.014	0	0	0	0	0.014
7004	Airboss of America Corporation	Kitchener	ON	0.013	0	0	0	0	0.013
0031	BASF Canada	Windsor	ON	0	0	0.009	0	0	0.009
4175	Schenectady Canada Ltd.	Toronto	ON	0.006	0.001	0	0	0	0.007
0222	Cytec Canada Inc.	Niagara Falls	ON	0.004	0	0	0	0	0.004
0249	Ashland Canada Corporation	Ajax	ON	0	0.001	0	0	0	0.001
5526	Société en commandite Tafisa Canada	Lac-Mégantic	QC	84.1	0	0	0	0	84.1
2989	Uniboard Canada inc.	Sayabec	QC	79.069	0	0.791	0	0	79.86
0758	Uniboard Canada Inc.	Mont-Laurier	QC	68.04	0	0	0	0	68.04
5518	Louisiana Pacific Canada Ltd	Bois-Franc	QC	66.006	0	0	0	0	66.006
1745	Industries Norbord Inc.	Val d'Or	QC	57.25	0	0	0	0	57.25
4060	Uniboard Canada Inc.	Val-d'Or	QC	55.02	0	0	0	0	55.02

Table A-2 Formaldehyde Air Emissions in Alberta According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by province and by total emissions) (continued)

NPRI ID	Company	City	Province	Formaldehyde Emissions (in tonnes)					
				Stack /Point	Storage /Handling	Fugitive	Spills	Other Non-Point	Total
5442	Uniboard Canada Inc.	La Baie	QC	52.78	0	0	0	0	52.78
1681	Dynea Canada Ltée	Ste Therese	QC	6.5	0.023	11.82	0	0	18.343
3242	SFK Pâte S.E.N.C	St-Félicien	QC	4.811	0	0.02	0	0	4.831
1748	Industries Norbord Inc.	La Sarre	QC	15.77	0	0	0	0	15.77
2948	Tembec Inc	Témiscaming	QC	2.98	0	0.008	0	0	2.988
5516	Louisiana Pacific Canada Ltd	Saint-Michel-des-Saints	QC	14.584	0	0	0	0	14.584
5515	KRUGER	Trois-Rivières	QC	13.492	0	0	0	0	13.492
0279	Domtar Inc.	Lebel-sur-Quévillon	QC	4.72	0.006	0	0	0	4.726
4068	Papier Stadacona Ltee	Québec	QC	0	0	1.615	0	0	1.615
4362	Isolation Manson inc.	Brossard	QC	10.6	0	0	0	0	10.6
1858	Owens-Corning Canada Inc.	Candiac	QC	7.174	0.026	0	0	0	7.2
0929	BOWATER	Gatineau	QC	2	0	0	0	0	2
6441	Uniboard Canada Inc	Laval	QC	2.57	1.03	0	0	0	3.6
2001	La Compagnie Abitibi-Consolidated du Canada	Baie-Comeau	QC	2.597	0	0	0	0	2.597
3381	Uniboard Canada Inc.	Val-d'Or	QC	3.87	0	0	0	0	3.87
4386	Tembec Inc.	St-Georges-de-Champlain	QC	2.9	0	0	0	0	2.9
0007	Borden Chimie Canada, Inc.	Laval	QC	1.136	0.238	0.18	0	0	1.554
4802	Produits Chimiques Handy Ltee	Candiac	QC	1.4	0	0	0	0	1.4
5429	Tembec Inc.	Longueuil	QC	1.3	0	0	0	0	1.3
5430	3io corp	Riviere Trois-Pistoles	QC	0.67	0	0	0	0	0.67
1648	Solutia Canada inc.	LaSalle	QC	0.203	0	0.106	0	0	0.309
6234	Fournitures Funéraires Victoriaville inc.	Victoriaville	QC	0.169	0	0	0	0	0.169
5449	Clean Harbors	Mercier	QC	0.021	0.141	0	0	0	0.162

Table A-2 Formaldehyde Air Emissions in Alberta According to the 2003 NPRI Database (EC, 2005) (in tonnes, ranked by province and by total emissions) (continued)

NPRI ID	Company	City	Province	Formaldehyde Emissions (in tonnes)					
				Stack /Point	Storage /Handling	Fugitive	Spills	Other Non-Point	Total
5455	Services environnementaux Clean Harbors Québec, Inc	Thurso	QC	0	0.154	0	0	0	0.154
3095	BAKOR INC.	Lachine	QC	0	0	0.15	0	0	0.15
4407	Arborite,division de ITW Canada	LaSalle	QC	0	0.147	0	0	0	0.147
5556	Borden Chimie Canada, Inc.	St-Romuald	QC	0.079	0.06	0.002	0	0	0.141
0330	Ferox Inc / Laques International Inc	Anjou	QC	0	0	0.1	0	0	0.1
3125	Société Viasystems Canada S.E.N.C.	Kirkland	QC	0.022	0	0	0	0	0.022
5454	Clean Harbors environmental services, Inc.	Ste-Catherine	QC	0	0	0.02	0	0	0.02
2334	Univar Canada Ltd.	Valleyfield	QC	0	0.007	0	0	0	0.007
20040	Meadow Lake OSB Limited Partnership	Meadow Lake	SK	16.367	0	0	0	0	16.367

APPENDIX B
Air Quality Guidelines for Formaldehyde Development and Use

Agency: Alberta Environment (AENV).
Air Quality Guideline: Ambient Air Quality Objective (AAQO) = 65 µg m^{-3}.
Averaging Time To Which Guideline Applies: 1-hour averaging time.
Basis for Development: Adopted from Texas (however, the Texas Commission on Environmental Quality has since revised their ESL, refer to the table for Texas for their latest ESLs).
Date Guideline Developed: 1999.
How Guideline is Used: Used by Alberta Environment to establish approval conditions and can be used to assess compliance and evaluate performance at industrial facilities.
Additional Comments:
Reference and Supporting Documentation: Alberta Environment. 2005. Alberta Ambient Air Quality Objectives. Alberta Environment, Environmental Policy Branch, Edmonton, AB. April 2005. 4 pp. http://www3.gov.ab.ca/env/protenf/approvals/factsheets/ABAmbientAirQuality.pdf (accessed 15 October 2005).

Agency: British Columbia Ministry of Environment (MOE).
Air Quality Guideline: Air Quality Objective (AQO): 60 $\mu g\ m^{-3}$ as an action level 370 $\mu g\ m^{-3}$ as an episode level
Averaging Time To Which Guideline Applies: 1-hour averaging time.
Basis for Development: Unknown.
Date Guideline Developed: 1995.
How Guideline is Used: British Columbia has a two-tiered objective comprising of an "action" level and an "episode" level. The action level is the target used when managing the level of formaldehyde in an airshed. The episode level corresponds to the concentration that starts to be of concern to public health (it is at this level that immediate steps be taken to reduce the release of formaldehyde into the atmosphere).
Additional Comments:
Reference and Supporting Documentation: British Columbia Ministry of Environment (MOE). 2005. Air Quality Objectives and Standards, Air Quality Objectives for British Columbia and Canada. British Columbia MOE, Victoria, BC. http://wlapwww.gov.bc.ca/air/airquality/pdfs/aqotable.pdf (accessed 15 October 2005).

Agency: Manitoba Conservation.
Air Quality Guideline: Ambient Air Quality Criteria – maximum acceptable level concentration: 60 $\mu g\ m^{-3}$.
Averaging Time To Which Guideline Applies: 1-hour averaging time.
Basis for Development: Unknown.
Date Guideline Developed: Unknown.
How Guideline is Used: Used by Manitoba Conservation to serve as a guide for the evaluation of air quality and for planning purposes.
Additional Comments: Maximum acceptable level concentrations are deemed by Manitoba Conservation as essential to provide adequate protection for soils, water, vegetation, materials, animals, visibility, personal comfort and well-being.
Reference and Supporting Documentation: Manitoba Conservation. 2005. Objectives and Guidelines for Various Air Pollutants: Ambient Air Quality Criteria (updated July, 2005). Manitoba Conservation, Air Quality Section, Winnipeg, MB. http://www.gov.mb.ca/conservation/airquality/aq-criteria/ambientair_e.html (accessed 15 October 2005).

Agency: Ontario Ministry of the Environment (OME).
Air Quality Guideline: ½-hour standard: 65 μg m^{-3} as a 30-minute average. 24-hour standard: 65 μg m^{-3} as a 24-hour average.
Averaging Time To Which Guideline Applies: Various averaging times indicated above.
Basis for Development: The ½-hour standard is based on odour/irritation and the 24-hour standard is based on health.
Date Guideline Developed: 2004
How Guideline is Used: The standards are used by Ontario Ministry of Environment (OME) to represent human health or environmental effect-based values not expected to cause adverse effects based on continuous exposure.
Additional Comments:
Reference and Supporting Documentation: Ontario Ministry of the Environment (OME). 2005. Summary of O. Reg. 419/05 Standards and Point of Impingement Guidelines and Ambient Air Quality Criteria (AAQCs). Standards Development Branch, Ontario Ministry of the Environment, Toronto, ON. December 2005. 16 pp. http://www.ene.gov.on.ca/envision/air/airquality/standards.htm (accessed 3 February 2006).

Agency:

US Agency for Toxic Substances and Disease Registry (ATSDR).

Air Quality Guideline:

Inhalation minimum risk levels (MRLs):

49 $\mu g\ m^{-3}$ (0.04 ppm) for the acute MRL

37 $\mu g\ m^{-3}$ (0.03 ppm) for the intermediate MRL

10 $\mu g\ m^{-3}$ (0.008 ppm) for the chronic MRL

Averaging Time To Which Guideline Applies:

≤14 days continuous exposure for the acute MRL

14 days to ≤365 days continuous exposure for the intermediate MRL

>365 days continuous exposure for the chronic MRL

Basis for Development:

The acute inhalation MRL was calculated based on a minimal LOAEL of 0.4 ppm for symptoms of increased itching, sneezing, mucosal congestion, and burning sensation of the eyes and nasal passages, and elevated eosinophil counts and a transient increase in albumin content of nasal lavage fluid in volunteers exposed to formaldehyde for 2 hours. The LOAEL was divided by an uncertainty factor of 9 (3 for the use of a minimal LOAEL and 3 for human variability).

An intermediate inhalation MRL of 37 $\mu g\ m^{-3}$ (0.03 ppm) was calculated based on a NOAEL of 0.98 ppm for nasopharyngeal irritation in Cynomolgus monkeys and using an uncertainty factor of 30 (3 for extrapolation from animals to humans and 10 for human variability).

A chronic inhalation MRL of 10 $\mu g\ m^{-3}$ (0.008 ppm) was calculated based on a minimal LOAEL of 0.24 ppm for mild nasal lesions in chemical factory workers and using an uncertainty factor of 30 (3 for the use of a minimal LOAEL and 10 for human variability).

Date Guideline Developed:

July 1999.

How Guideline is Used:

MRLs are intended to serve as a screening tool to help public health professionals decide where to look more closely. Inhalation MRLs are exposure concentrations that, based on current information, might cause adverse health effects in the people most sensitive to such substance-induced effects for exposure durations described above.

Additional Comments:

Inhalation MRLs provide a basis for comparison with levels that people might encounter in air. If a person is exposed to formaldehyde at an amount below the MRL, it is not expected that harmful (noncancer) health effects will occur. Because these levels are based only on information currently available, some uncertainty is always associated with them. Also, because the method for deriving MRLs does not use any information about cancer, an MRL does not imply anything about the presence, absence, or level of risk for cancer.

Reference and Supporting Documentation:

Agency for Toxic Substances and Disease Registry (ATSDR). 2005. Minimal Risk Levels (MRLs) for Hazardous Substances. ATSDR, Public Health Service, US Department of Health and Human Services. Atlanta, GA. http://www.atsdr.cdc.gov/mrls.html (accessed 15 Oct 2005).

Agency: US Environmental Protection Agency (EPA).
Air Quality Guideline: Risk specific Concentration (RsC) corresponding to 1 in 100,000 risk = 0.8 μg m^{-3}.
Averaging Time To Which Guideline Applies: Continuous exposure (daily exposure over a lifetime).
Basis for Development: The RsC corresponding to 1 in 100,000 risk (risk criteria used in Alberta) was derived based on observation of squamous cell carcinoma in a 2-year rat study and using the linearized multistage procedure.
Date Guideline Developed: 1989.
How Guideline is Used: The risk specific concentration (RsC) is not used for any specific purposes by US EPA and is shown here to illustrate an exposure concentration in air associated with an inhalation unit risk factor derived by US EPA and a 1 in 100,000 lifetime cancer risk.
Additional Comments: The Integrated Risk Information System (IRIS) is prepared and maintained by the US EPA. IRIS is an electronic database containing information on human health effects that may result from exposure to various chemicals in the environment.
Reference and Supporting Documentation: US Environmental Protection Agency. 2005. Integrated Risk Information System. http://www.epa.gov/iris/ (accessed 15 October 2005).

Agency:

Arizona Department of Environmental Quality (DEQ).

Air Quality Guideline:

Arizona Ambient Air Quality Guidelines (AAAQGs):

Annual AAAQG: 0.08 $\mu g\ m^{-3}$

24-hour AAAQG: 12 $\mu g\ m^{-3}$

1-hour AAAQG: 20 $\mu g\ m^{-3}$

Averaging Time To Which Guideline Applies:

See above.

Basis for Development:

The annual AAAQG is the US Environmental Protection Agency (2005) Risk specific Concentration (RsC) corresponding to 1 in 1,000,000 risk (10^{-6}).

The derivation basis for the 24-hour AAAQG is unknown.

The 1-hour AAAQG is derived by taking the US Occupational Safety and Health Administration (OSHA) 15-minute Short Term Exposure Limit (STEL) of 2 ppm (2,456 $\mu g\ m^{-3}$) and dividing it by a factor of 120 and rounded down. The factor of 120 converts a 15-minute exposure into a one-hour exposure, and a safety factor of 30 to protect the most sensitive members of the population such as children and the elderly.

Date Guideline Developed:

Unknown.

How Guideline is Used:

AAAQGs are used by Arizona DEQ to review permit applications for formaldehyde sources and as criteria to investigate complaints and violations of Arizona's air quality laws. The Arizona Ambient Air Quality Guidelines (AAAQG) are acceptable concentration levels for hazardous air pollutants that are regulated by the State of Arizona.

Additional Comments:

Reference and Supporting Documentation:

Arizona Department of Environmental Quality (DEQ). 2005. Arizona Ambient Air Quality Guidelines. Arizona DEQ, Air Quality Division, Phoenix, AZ. 10 pp.
http://www.azdeq.gov/environ/air/index.html (accessed 15 October 2005).

Arizona Department of Health Services (DHS). 1999. 1999 Update – Arizona Ambient Air Quality Guidelines (AAAQGs). Report prepared for Arizona Department of Environmental Quality, Air Programs Division. Arizona DHS, Office of Environmental Health, Phoenix, AZ. 11 May 1999. 20 pp.

US Environmental Protection Agency. 2005. Integrated Risk Information System. http://www.epa.gov/iris/ (accessed 15 October 2005).

Agency:

California Environmental Protection Agency (Cal EPA).

Air Quality Guideline:

Acute reference exposure level (REL) = 94 $\mu g\ m^{-3}$

Chronic reference exposure level (REL) = 3 $\mu g\ m^{-3}$

Averaging Time To Which Guideline Applies:

Acute REL: 1-hour averaging time.

Chronic REL: continuous (daily) exposure over a lifetime.

Basis for Development:

Acute REL: The acute REL was based on a benchmark concentration (BC_{05}) approach, using log-probit analysis. The BC_{05} is defined as the 95% lower confidence limit of the concentration expected to produce a response rate of 5%. The resulting BC_{05} from the analysis was 0.44 ppm (0.53 mg m^{-3}) formaldehyde. This value was adjusted to a 1-hour duration of 0.74 ppm. This was converted to 0.94 mg m^{-3} using the Cal EPA factor of 1 ppm = 1.24 mg m^{-3} at 25°C. An uncertainty factor (UF) of 10 was used to account for individual variation.

Chronic REL: The chronic REL is based on discontinuous occupational exposure in 66 human chemical plant workers. The average occupational concentration of 0.032 mg m^{-3} was estimated for a NOAEL and a cumulative uncertainty factor of 10 was applied.

Date Guidelines Developed:

Unknown.

How Guideline is Used:

Acute and chronic RELs are for use in facility health risk assessments conducted for the AB 2588 Air Toxics "Hot Spots" Program.

Additional Comments:

Reference and Supporting Documentation:

California Office of Environmental Health Hazard Assessment (OEHHA). 1999. Acute Toxicity Summary: Formaldehyde. Cal OEHHA, Sacramento, CA. 8 pp. http://www.oehha.ca.gov/air/acute_rels/allAcRELs.html (accessed 15 October 2005).

California Office of Environmental Health Hazard Assessment (OEHHA). 2003. Chronic Toxicity Summary: Fluorides including Formaldehyde. Cal OEHHA, Sacramento, CA. 16 pp. http://www.oehha.ca.gov/air/chronic_rels/AllChrels.html (accessed 15 October 2005).

Agency: Indiana Department of Environmental Management (IDEM).
Air Quality Guideline: IDEM does not have an air quality guideline for this chemical.
Averaging Time To Which Guideline Applies:
Basis for Development:
Date Guideline Developed:
How Guideline is Used:
Additional Comments:
Reference and Supporting Documentation: Indiana Department of Environmental Management (DEM). 2002. Office of Air Quality Programs. Indiana DEM, Office of Air Quality. Indianapolis, IN. http://www.in.gov/idem/air/programs/modeling/policy.html (accessed 15 October 2005).

Agency: Louisiana Department of Environmental Quality (DEQ).
Air Quality Guideline: Ambient air standard (AAS) for toxic air pollutants = 7.69 μg m^{-3}.
Averaging Time To Which Guideline Applies: Annual averaging time.
Basis for Development: Unknown.
Date Guideline Developed: December 2003.
How Guideline is Used: AASs are used by Louisiana DEQ to review permit applications for stationary sources that emit formaldehyde to the atmosphere.
Additional Comments:
Reference and Supporting Documentation: Louisiana Department of Environmental Quality (DEQ). 2003. Louisiana Administrative Code (LAC). Title 33 Environmental Quality, Part III Air, Chapter 51. Comprehensive Toxic Air Pollutant Emission Control Program. Louisiana Department of Environmental Quality. Baton Rouge, LA. http://www.state.la.us/osr/lac/lac33.htm (accessed 15 October 2005).

Agency:

Massachusetts Department of Environmental Protection (DEP).

Air Quality Guideline:

Threshold Effects Exposure Limit (TEL) = 0.33 $\mu g\ m^{-3}$ as a 24-hour averaging time.

Allowable Ambient Limit (AAL) = 0.08 $\mu g/m^3$ as an annual average.

Averaging Time To Which Guideline Applies:

See above.

Basis for Development:

The derivation basis for the Threshold Effects Exposure Limit (TEL) is unknown.

The Allowable Ambient Limit (AAL) is the US Environmental Protection Agency (2005) Risk specific Concentration (RsC) corresponding to 1 in 1,000,000 risk (10^{-6}).

Date Guideline Developed:

Unknown.

How Guideline is Used:

Information could not be obtained to identify how the guideline is used, but it is expected that the guideline is used in some manner to meet state level permitting.

Additional Comments:

Reference and Supporting Documentation:

Massachusetts Department of Environmental Protection (DEP). 1995. Revised air guidelines [updated list of 24-hour average Threshold Effects Exposure Limit (TEL) values and annual average Allowable Ambient Limit (AAL) values]. Memorandum. Massachusetts DEP, Boston, MA. 6 December 1995. http://www.mass.gov/dep/air/aallist.pdf (accessed 15 October 2005).

Massachusetts Department of Environmental Protection (DEP). 1990. Chemical Health Effects Assessment Methodology & Method to Derive Allowable Ambient Limits (CHEM/AAL). Massachusetts DEP, Boston, MA. February 1990. http://www.mass.gov/dep/air/laws/policies.htm (accessed 15 October 2005).

Agency: Michigan Department of Environmental Quality (DEQ).
Air Quality Guideline: Initial risk screening level (IRSL) = 0.08 $\mu g\ m^{-3}$. Secondary risk screening level (SRSL) = 0.8 $\mu g\ m^{-3}$.
Averaging Time To Which Guideline Applies: Annual averaging time.
Basis for Development: The IRSL is the US Environmental Protection Agency (2005) Risk specific Concentration (RsC) corresponding to 1 in 1,000,000 risk (10^{-6}). The SRSL is the US Environmental Protection Agency (2005) Risk specific Concentration (RsC) corresponding to 1 in 100,000 risk (10^{-5}).
Date Guideline Developed: 1988.
How Guideline is Used: There are two basic requirements of Michigan air toxic rules. First, each source must apply the best available control technology for toxics (T-BACT). After the application of T-BACT, the emissions of the toxic air contaminant cannot result in a maximum ambient concentration that exceeds the applicable health based screening level for non-carcinogenic effects (ITSL). Application of an ITSL is required for any new or modified emission source or sources for which a permit to install is requested and which emits a toxic air contaminant.
Additional Comments: The applicable air quality screening level for chemical treated as non-carcinogens by Michigan DEQ is the ITSL. There are two health based screening levels for chemical treated as carcinogens by Michigan DEQ: the initial risk screening level (IRSL) – based on an increased cancer risk of one in one million, and the secondary risk screening level (SRSL) – based on as an increased cancer risk of 1 in 100,000.
Reference and Supporting Documentation: Michigan Department of Environmental Quality (DEQ). 2005. Initial Threshold Screening Level (ITSL) / Initial Risk Screening Level (IRSL) Glossary. Michigan DEQ, Air Quality division, Lansing, MI. http://www.michigan.gov/deq/0,1607,7-135-3310_4105-11754--,00.html (accessed 8 November 2005). Michigan Department of Environmental Quality (DEQ). 1998. Michigan Administrative Code (MAC). Air Pollution Control Rules. Part 2 Air Use Approval, R 336.1201 - 336.1299. Air Quality Division, Department of Environmental Quality. Lansing, MI. http://www.state.mi.us/orr/emi/admincode.asp?admincode=Department&Dpt=EQ (accessed 8 November 2005).

Agency:

Minnesota Department of Health (DOH).

Air Quality Guideline:

Acute Health Risk Value (HRV) = 94 $\mu g\ m^{-3}$.

Chronic Health Risk Value (HRV) = 0.8 $\mu g\ m^{-3}$.

Averaging Time To Which Guideline Applies:

Acute HRV: 1-hour averaging time.

Chronic HRV: annual averaging time.

Basis for Development:

The acute HRV for formaldehyde was adopted directly from the California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, Acute reference exposure level (REL) of 94 $\mu g\ m^{-3}$.

The chronic HRV for formaldehyde is the US Environmental Protection Agency (2005) Risk specific Concentration (RsC) corresponding to 1 in 100,000 risk (10^{-5}).

Date Guideline Developed:

March 2002.

How Guideline is Used:

HRVs are used by the Minnesota Department of Health and sister agencies such as the Minnesota Pollution Control Agency, to assist in the assessment of potential health risks associated with chemicals in ambient air. HRVs can be used as one set of criteria for assessing risks in the environmental review process, issuing air permits, risk assessments and other site-specific assessments.

Additional Comments:

The Inhalation Health Risk Values are "concentrations of chemicals or substances in the air that are estimated to produce no significant increased risk of harmful effects for specific lengths of exposure."

Reference and Supporting Documentation:

Minnesota Department of Health (DOH). 2005. Health Risk Values for Air. Minnesota DOH, St. Paul, MN. http://www.health.state.mn.us/divs/eh/air/hrvtable.htm#chronic (accessed 15 November 2005).

Agency: New Hampshire Department of Environmental Services (DES).
Air Quality Guideline: 24-hour ambient air limit (AAL) = 1.3 $\mu g\ m^{-3}$. Annual ambient air limit (AAL) = 0.88 $\mu g\ m^{-3}$.
Averaging Time To Which Guideline Applies: See above.
Basis for Development: 24-hour Ambient Air Limit – Unknown. Annual Ambient Air Limit – Unknown.
Date Guideline Developed: Unknown.
How Guideline is Used: AALs are used by New Hampshire DES to review permit applications for sources that emit formaldehyde to the atmosphere. Sources are regulated through a statewide air permitting system and include any new, modified or existing stationary source, area source or device.
Additional Comments:
Reference and Supporting Documentation: New Hampshire Department of Environmental Services (DES). 2005. New Hampshire Administrative Rule. Chapter Env-A 1400. Regulated Toxic Air Pollutants. New Hampshire Department of Environmental Services. Concord, NH. http://www.des.state.nh.us/rules/env-a1400.pdf (accessed 15 October 2005).

Agency: New Jersey Department of Environmental Protection (DEP).
Air Quality Guideline: Applicants are required to carry out a risk assessment in conjunction with applying for an air pollution control pre-construction permit. In the case of formaldehyde, the US Environmental Protection Agency inhalation unit risk factor of 1.3E-05 per $\mu g\ m^{-3}$ is used to calculate a lifetime cancer risk for sources that emit formaldehyde to the atmosphere.
Averaging Time To Which Guideline Applies: Continuous exposure (daily exposure over a lifetime).
Basis for Development: Based on US EPA Integrated Risk Information System (IRIS) data.
Date Guideline Developed: December 1994.
How Guideline is Used:
Additional Comments:
Reference and Supporting Documentation: New Jersey Department of Environmental Protection (DEP). 2005. New Jersey Administrative Code (NJAC). Title 7, Chapter 27, Subchapter 8. Permits and Certificates for Minor Facilities (and Major Facilities without an Operating Permit). New Jersey Department of Environmental Protection. Trenton, NJ. http://www.state.nj.us/dep/aqm/rules.htm (accessed 15 October 2005). New Jersey Department of Environmental Protection (DEP). 1994. Technical Manual 1003. Guidance on Preparing a Risk Assessment for Air Contaminant Emissions. Air Quality Permitting Program, Bureau of Air Quality Evaluation, New Jersey DEP, Trenton, NJ. Revised December 1994. http://www.state.nj.us/dep/aqpp/downloads/techman/1003.pdf (accessed 15 October 2005).

Agency: North Carolina Department of Environment and Natural Resources (ENR).
Air Quality Guideline: Acceptable ambient level (AAL): 150 $\mu g\ m^{-3}$.
Averaging Time To Which Guideline Applies: 1-hour averaging time.
Basis for Development: Unknown
Date Guideline Developed: Unknown.
How Guideline is Used: A facility emitting formaldehyde must limit its emissions so that the resulting modeled ambient levels at the property boundary remain below the health-based acceptable ambient level (AAL).
Additional Comments:
Reference and Supporting Documentation: North Carolina Department of Environment and Natural Resources. 2005. North Carolina Administrative Code (NCAC). North Carolina Air Quality Rules 15A NCAC 2D.1100 – Air Pollution Control Requirements (Control of Toxic Air Pollutants). North Carolina Department of Environment and Natural Resources, Raleigh, NC. http://reports.oah.state.nc.us/ncac.asp (accessed 15 October 2005).

Agency: Ohio Environmental Protection Agency (EPA).
Air Quality Guideline: Ohio EPA does not have an air quality guideline for this chemical.
Averaging Time To Which Guideline Applies:
Basis for Development:
Date Guideline Developed:
How Guideline is Used:
Additional Comments:
Reference and Supporting Documentation: Ohio Environmental Protection Agency (EPA). 2005. Air Toxics Policy – Option A: Review of New Sources of Toxic Emissions. Air Toxics Unit, Division of Air Pollution Control, Ohio EPA. Columbus, OH. 11 pp. http://www.epa.state.oh.us/dapc/atu/atu.html (accessed 15 October 2005). Ohio Environmental Protection Agency (Ohio EPA). 1994. Review of New Sources of Air Toxic Emissions. Proposed for Public Comment. Division of Air Pollution Control, Ohio EPA. Columbus, OH. January 1994. 31 pp. http://www.epa.state.oh.us/dapc/atu/atu.html (accessed 15 October 2005).

Agency: Oklahoma Department of Environmental Quality (DEQ).
Air Quality Guideline: Maximum acceptable ambient concentration (MAAC) =12 µg m^{-3}.
Averaging Time To Which Guideline Applies: 24-hour averaging time.
Basis for Development: Unknown.
Date Guideline Developed: Not stated.
How Guideline is Used: MAACs are used by Oklahoma DEQ to review permit applications for sources that emit molybdenum to the atmosphere.
Additional Comments:
Reference and Supporting Documentation: Oklahoma Department of Environmental Quality (DEQ). 2005. Oklahoma Administrative Code (OAC). Title 252. Chapter 100. Air Pollution Control. 100:252-41 - Control of Emission of Hazardous and Toxic Air Contaminants. Oklahoma DEQ, Oklahoma City, OK. http://www.sos.state.ok.us/oar/oar_welcome.htm (accessed 15 October 2005). Oklahoma Department of Environmental Quality (DEQ). 2002. Air Toxics Partial Listing [maximum acceptable ambient concentrations (MAAC) for air toxics]. Oklahoma City, OK. http://www.deq.state.ok.us/AQDNew/toxics/listings/pollutant_query_1.html (accessed 15 October 2005).

Agency: Pennsylvania Department of Environmental Protection (DEP).
Air Quality Guideline: Pennsylvania DEP does not have an air quality guideline for this chemical.
Averaging Time To Which Guideline Applies:
Basis for Development:
Date Guideline Developed:
How Guideline is Used:
Additional Comments:
Reference and Supporting Documentation: Pennsylvania Department of Environmental Protection (DEP). 2005. Pennsylvania State Code, Article III Air Resources, Section 131.1, Ambient Air Quality Standards. Pennsylvania DEP, Bureau of Air Quality, Harrisburg, PA, http://www.pacode.com/secure/data/025/articleICIII_toc.html (accessed 15 October 2005).

Agency:

Rhode Island Department of Environmental Management (DEM).

Air Quality Guideline:

Acceptable ambient level (AAL) for formaldehyde (proposed):

1-hour AAL – 50 $\mu g\ m^{-3}$

24-hour AAL – 40 $\mu g\ m^{-3}$

Annual AAL – 0.08 $\mu g\ m^{-3}$

Averaging Time To Which Guideline Applies:

See above.

Basis for Development:

The proposed 1-hour AAL is based on the US Agency for Toxic Substances and Disease Registry (ATSDR) acute inhalation MRL rounded to 50 $\mu g\ m^{-3}$.

The derivation basis for the proposed 24-hour AAL is unknown.

The proposed annual AAL is the US Environmental Protection Agency (2005) Risk specific Concentration (RsC) corresponding to 1 in 1,000,000 risk (10^{-6}).

Date Guideline Developed:

April 2004.

How Guideline is Used:

AALs are used by Rhode Island DEM to review permit applications for sources that emit manganese to the atmosphere.

Additional Comments:

Reference and Supporting Documentation:

Rhode Island Department of Environmental Management. 2004. Air Pollution Control Regulation #22, Air Toxics. Division of Air and Hazardous Materials, Rhode Island Department of Environmental Management. Providence, RI. Amended 27 April 2004. http://www.state.ri.us/dem/pubs/regs/index.htm#Air (accessed 15 October 2005).

Agency: Texas Commission on Environmental Quality (CEQ) – formerly Texas Natural Resource Conservation Commission (TRNCC).
Air Quality Guideline: Short-term effects screening level (ESL) = 15 $\mu g\ m^{-3}$. Long-term effects screening level (ESL) = 1.5 $\mu g\ m^{-3}$.
Averaging Time To Which Guideline Applies: 1-hour averaging time for short-term ESL. Annual averaging time for long-term ESL.
Basis for Development: Short-term Effects Screening Level – Unknown. Long-term Effects Screening Level – Unknown.
Date Guideline Developed: Not stated.
How Guideline is Used: ESLs are used to evaluate the potential for effects to occur as a result of exposure to concentrations of constituents in air. ESLs are based on data concerning health effects, odor nuisance potential, effects with respect to vegetation, and corrosion effects. They are not ambient air standards. If predicted or measured airborne levels of a chemical do not exceed the screening level, adverse health or welfare effects would not be expected to result. If ambient levels of constituents in air exceed the screening levels, it does not necessarily indicate a problem, but rather, triggers a more in-depth review.
Additional Comments:
Reference and Supporting Documentation: Texas Commission on Environmental Quality (CEQ). 2003. Effects Screening Levels. TCEQ Toxicology Section, Austin, TX. http://www.tceq.state.tx.us/implementation/tox/esl/list_main.html (accessed 15 October 2005).

Agency: Vermont Agency of Natural Resources (ANR).
Air Quality Guideline: Hazardous ambient air standard (HAAS) = 0.08 $\mu g\ m^{-3}$.
Averaging Time To Which Guideline Applies: Annual averaging time.
Basis for Development: The HAAS is the US Environmental Protection Agency (2005) Risk specific Concentration (RsC) corresponding to 1 in 1,000,000 risk (10^{-6}).
Date Guideline Developed: Not stated.
How Guideline is Used: HAASs are used by Vermont ANR to review permit applications for stationary sources that emit formaldehyde to the atmosphere.
Additional Comments:
Reference and Supporting Documentation: Vermont Agency of Natural Resources (ANR). 2001. Air Pollution Control Regulations. State of Vermont Agency of Natural Resources, Air Pollution Control Division, Waterbury, VT. http://www.anr.state.vt.us/air/AirToxics/docs/apcregs.pdf (accessed 15 October 2005).

Agency: Washington State Department of Ecology (DOE).
Air Quality Guideline: Acceptable source impact level (ASIL) = 0.077 μg m^{-3}.
Averaging Time To Which Guideline Applies: Annual averaging time.
Basis for Development: The ASIL is the US Environmental Protection Agency (2005) Risk specific Concentration (RsC) corresponding to 1 in 1,000,000 risk (10^{-6}).
Date Guideline Developed: Unknown.
How Guideline is Used: ASILs are used by Washington State DOE to review permit applications for sources that emit formaldehyde to the atmosphere.
Additional Comments:
Reference and Supporting Documentation: Washington State Department of Ecology (DOE). 2005. Washington Administrative Code (WAC). Chapter 173-460 WAC. Controls For New Sources Of Toxic Air Pollutants. Washington State DOE, Olympia, WA. http://www.leg.wa.gov/wac/ (accessed 15 October 2005).

Agency: Wisconsin Department of Natural Resources (DNR).
Air Quality Guideline: Wisconsin DNR does not have an air quality guideline for this chemical.
Averaging Time To Which Guideline Applies:
Basis for Development:
Date Guideline Developed:
How Guideline is Used:
Additional Comments:
Reference and Supporting Documentation: Wisconsin Department of Natural Resources (DNR). 2005. Wisconsin Administrative Code (WAC). Air Pollution Control Rules. Chapter NR 445. Control of Hazardous Pollutants. Wisconsin DNR, Madison WI. http://www.legis.state.wi.us/rsb/code/nr/nr445.pdf (accessed 15 October 2005).

Agency: New Zealand Ministry for the Environment (MOE) and New Zealand Ministry of Health (MOH).
Air Quality Guideline: Health-based guideline value for formaldehyde: 30-minute averaging time: 100 μg m^{-3}.
Averaging Time To Which Guideline Applies: See above.
Basis for Development: This guideline value is based on the WHO (2000) value and is designed to protect most individuals.
Date Guideline Developed: 2002.
How Guideline is Used: Health-based guideline values are intended to be used by the New Zealand MOE and MOH to direct air-shed management and evaluate ambient air quality monitoring results.
Additional Comments:
Reference and Supporting Documentation: New Zealand Ministry for the Environment and Ministry of Health (New Zealand). 2002. Ambient Air Quality Guidelines 2002 Update. Prepared by the Ministry for the Environment and the Ministry of Health. Wellington, New Zealand. May 2002. 58 pp. www.mfe.govt.nz (accessed 3 February 2006).

Agency: The Netherlands National Institute of Public Health and the Environment (RIVM)
Air Quality Guideline: RIVM does not have air quality criteria for formaldehyde.
Averaging Time To Which Guideline Applies:
Basis for Development:
Date Guideline Developed:
How Guideline is Used:
Additional Comments:
Reference and Supporting Documentation: The Netherlands National Institute of Public Health and the Environment (RIVM). 2001. Re-evaluation of human-toxicological maximum permissible risk levels. RIVN Report 711701 025. RIVN, Bilthoven, The Netherlands. March 2001. 297 pp.

Agency:

Health Canada.

Air Quality Guideline:

Proposed residential indoor air quality guideline for formaldehyde:

123 $\mu g\ m^{-3}$ as an 1-hour (short-term) guideline

50 $\mu g\ m^{-3}$ as an 8-hour (long-term) guideline

Averaging Time To Which Guideline Applies:

Refer to above.

Basis for Development:

Unknown.

Date Guideline Developed:

August 9, 2005.

How Guideline is Used:

Used as an indoor guideline.

Additional Comments:

Studies carried out in Canada since the early 1990s consistently indicate that formaldehyde concentrations in Canadian homes range between 2.5 and 88 $\mu g\ m^{-3}$ with an average between 30 and 40 $\mu g\ m^{-3}$.

Reference and Supporting Documentation:

Health Canada. 2005. Proposed residential indoor air quality guideline for formaldehyde. Health Canada, Ottawa, ON. http://canadagazette.gc.ca/partI/2005/20050820/html/notice-e.html#i3 (accessed 15 October 2005).

Agency: World Health Organization (WHO).
Air Quality Guideline: Ambient air reference exposure level for general population = 100 μg m^{-3}.
Averaging Time To Which Guideline Applies: 30-minute averaging time.
Basis for Development: The lowest concentration that is report to be associated with nose and throat irritation in humans after short-term exposure by WHO (2000) is 0.1 mg m^{-3}, although WHO (2000) reported that some individuals can sense the presence of formaldehyde at lower concentrations. To prevent significant sensory irritation in the general population, an air quality guideline value of 0.1 mg m^{-3} (100 μg m^{-3}) as a 30-minute average was recommended by WHO (2000). WHO (2000) further stated that since this level is over one order of magnitude lower than a presumed threshold for cytotoxic damage to the nasal mucosa, it represents an exposure level at which there is a negligible risk of upper respiratory tract cancer in humans.
Date Guideline Developed: 2000.
How Guideline is Used: The guideline is intended to provide background information and guidance to governments in making risk management decisions, particularly in setting standards.
Additional Comments:
Reference and Supporting Documentation: World Health Organization (WHO). 2000. Air Quality Guidelines for Europe, 2nd Edition. WHO Regional Publications, European Series, No. 91. WHO Regional Office for Europe, Copenhagen. 273 pp.